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Once again, controlled sensitivity studies have demonstrated the efficacy of CHLOROMYCETIN. In one long-term study,[1] designed to eliminate variable factors in patterns of bacterial resistance, 5,600 consecutive cultures of gram-positive organisms were tested over a 16-month period. Of the four broad-spectrum antibiotics evaluated, CHLOROMYCETIN was consistently superior.

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Editorial

Where There Is No Vision—

Are good football players more important to our state than intelligent well trained physicians? It would seem so, or at least circumstance would warrant this assumption. In our state youngsters talented toward athletics have a far easier time financing their education that those seemingly gifted toward the profession of medicine; many liberal scholarships are offered in athletics, but a student who aspires to a career in medicine finds the financial door to his ambition almost closed. At the present the medical school has but two $500.00 tuitional scholarships which they can offer to outstanding freshmen student applicants. How much money there is available for the training of students in football is anyone's guess!

The majority of freshmen students in the medical school must depend upon parental aid in order to finance their way through their training. Lacking this, their only alternative is to borrow money. As a consequence most medical students are burdened with comparatively large interest carrying debts upon graduation and which they must pay back when they enter into practice. Most medical students are compelled to borrow because the majority of them comes from families of rather slender financial means; a recent survey revealed that the parents of most medical students have an income of less than $8,000.00 a year, a sum which does not permit much latitude in financing a medical education for an ambitious son or daughter.

Our neighbor states, Kansas and Texas, appear to manifest a better sense of values than we exhibit so far as encouraging superior students into the career of medicine. Their colleges not only seem able to afford the luxury of good football teams but they appear enlightened enough to see that substantial scholarships are available for promising students who hope to become doctors. Because they attract good students into their schools the quality of medical care which their future citizens will enjoy is being upgraded.

Last year the Oklahoma School of Medicine to meet its class quota for freshmen medical students was forced to accept all but five of the qualified student applicants; some had only minimal qualifications for entrance. Unfortunately, the admission board was compelled to deny a number of extremely desirable applicants admission because these students could not obtain the necessary money required for a tuitional fee. As a result, although all these students were citizens of our state they were compelled to enroll into adjacent state schools wherein scholarships were available. It may be reasonably predicted that those students who left our state because of insufficient funds and had to go to other states to obtain their professional education will in all likelihood remain in those states for subsequent practice. Can the State of Oklahoma afford this loss?

Another aspect to this whole problem of scholarships may be noted. Top students of highschool classes who desire to enter into the various science vocations—chemistry, physics and even teaching—have a multiplicity of financial sources to whom they may apply for help: The National Science Foundation, National Institute of Health, National Defense Organization and many others, the list is long! As a result the number of admissions into these vocations has increased fourfold. Medicine, on the other hand is losing ground so far as the number of desirable applicants to the profession. Each year, although there is a demand for more physicians and the demand is becoming more critical each year, the list of students who choose a medical career is decreasing. The chances are that this is because the financial burden imposed upon students for them to obtain a medical education has become too great. A strange paradox; medicine for long has had the repute of being the most remunerative of the profession. It would appear in our society wherein so much stress is placed on money as being

the measure of success that our medical school would be overwhelmed with applicants rather than having to "scrape the barrel" as they do today. But admission boards have no alternative, they must accept students of a lesser degree of intelligence in order to meet the student quota set for the school.

Indeed, this is a lamentable commentary on our society—one where not scholastic superiority determines whether a student becomes a physician but primarily does he have the money to pay for his education!

One can be sure when these facts become generally understood by the public at large the reaction aroused may be predicted "let the government take over; why not subsidize the education of medical students by the government?" And if this situation continues this is what will actually occur, the government will subsidize medical education. Yet, every thinking person realizes that if the government controls medical education obviously they must control medical practice. Is this the backdoor route to complete socialization of the profession? It seems likely!

How to correct the problem? The only solution is for private enterprise to make available more scholarship funds in order to attract students of excellence into the profession. If this be accomplished our medical school will be in a position to select a better quality of student to fill the class rolls.

Recently a committee from the Faculty Association of the Medical School discussed this problem and came to the realization of how critical it was. A foundation plan was proposed and first steps were taken toward its organization. This foundation is a means whereby contributions from private sources may be accepted to provide more medical scholarships for freshmen students. If you feel inclined to help toward a solution of this problem may I suggest you write to Doctor Leonard P. Eliel of the Faculty Association at the Research Foundation for particulars. Possibly you may be even inclined to contribute something now, even a token amount would be helpful. If every doctor in the state would contribute one dollar a year it would be sufficient to make up a fund for three or four more exceptional students to be added to the next year class. The Faculty Association hopes by the beginning of the next scholastic year to have as much as $2,000 available for this purpose.

Are you convinced that good practitioners of medicine are as important to our state as football players? A small contribution, even a token amount, to this fund would give proof of your conviction.—*D.W.B.*

For Doctors Only?

Over the years, physicians have been contacted by various insurance companies in regard to insurance programs fraudulently proclaiming or implying association endorsement. Such statements as "Sold Only to Members of the Oklahoma State Medical Association" or, "For Doctors Only" mislead the physician-buyer into believing that he is enjoying special rates and privileges. The insurance itself may be good or bad, depending upon the underwriting company, but it cannot compete dollar for dollar with the bona-fide group programs established and administered by the association.

Currently, health and accident insurance is offered through the North American Accident Insurance Company and life insurance through the Massachusetts Mutual Life Insurance Company. The former is administered through the Insurance Committee and the latter through the Oklahoma State Medical Association Insurance Trust, a group comprised of the association President, President-Elect and Executive Secretary.

Both of the plans were designed to offer group-rated premiums, stability and benefits tailored to the needs of of most physicians.

Individual policies may suit a specific instance as well as the group plans, but don't be misled by misrepresentative claims of association endorsement.

Scientific Articles

A Symposium

Growth Failure in Infancy and Childhood*

PROGRAM

Introduction—Harris D. Riley, Jr., M.D., *Professor of Pediatrics*

Endocrine Aspects—Doman K. Keele, M.D., *Assistant Professor of Pediatrics*

Metabolic Aspects—H. Neil Kirkman, M.D., *Assistant Professor of Pediatrics*

Neurological Aspects—Lucius Waites, M.D., *Assistant Professor of Pediatrics and Neurology*

Psychologic, Sensory and Perceptual Aspects—Sylvia Richardson, M.D., *Assistant Professor of Pediatrics*

Cardiac Aspects—Glen G. Cayler, M.D., *Assistant Professor of Pediatrics*

Infectious Aspects—Harris D. Riley, Jr., M.D., *Professor of Pediatrics*

INTRODUCTION: Doctor Riley

THE MAIN BUSINESS of a child is healthy growth and development. Because many disease processes affect them adversely, growth failure may furnish the first clue that disease is present. True and suspected growth failure are among the most frequent problems encountered by the practitioner who sees children.

When presented with a patient with growth failure, the physician must first determine the validity of the diagnosis. A large percentage of the children whom the average pediatrician sees because of this complaint have nothing wrong with them. Parents do not generally appreciate the normal slowing of fetal growth and other physiologic aspects of normal growth. Norms for physical measurement such as height and weight are easily obtained by lay persons and applied anxiously to their children without the realization that these are averages which do not allow for normal deviation. Only the physician can determine if the complaint of growth failure is valid.

The so-called average height or weight value for a particular age is a misleading index since it is usually only a lucky coincidence that the child's height or weight will match the average figure. Far more adequate is the percentile chart which is derived from the normal distribution bell-shaped curve. In percentile charts, the mean is the fiftieth percentile; the mean plus and minus two standard deviations respectively generally covers the biologic variation from the 3rd to the 97th of random sampling. While an individual whose measurements fall within these limits may be affected by disease, most measurements included within these two limits are generally considered statistically within normal range. Such charts clearly show the wide spread between children whose weight or height is low normal and those children in whom height or weight is high normal. Growth can be regarded as abnormal in most cases when repeated measurements are found to be outside of these limits. Most healthy children tend to follow the same growth channel as they grow older.

In order to utilize adequately such percentil charts, it is highly desirable to record serial measurements made at intervale appropriate to the child's age rather than at-

*From the Department of Pediatrics and the Children's Memorial Hospital, University of Oklahoma School of Medicine, Oklahoma City. Presented at the Dedicatory Meeting, Mabee Children's Hospital, Tulsa, Oklahoma, September 19, 1959.

tempt to use a single set of physical measurements which may be misleading. Figure 1 portrays the erroneous impression which may result from using only a single weight measurement. At 9½ months of age, both Patients A and B weighed essentially the same. However, it is very obvious that Patient B, who weighed only four pounds, two ounces, at birth, has demonstrated an excellent growth response and is now within normal percentile channels. On the other hand, Patient A, who weighed eight pounds at birth, has not gained adequately since six months and is an example of growth failure although his weight at 9½ months was the same as Patient B. The cause of the growth failure was found to be pyelonephritis superimposed on an obstructive lesion of the urinary tract.

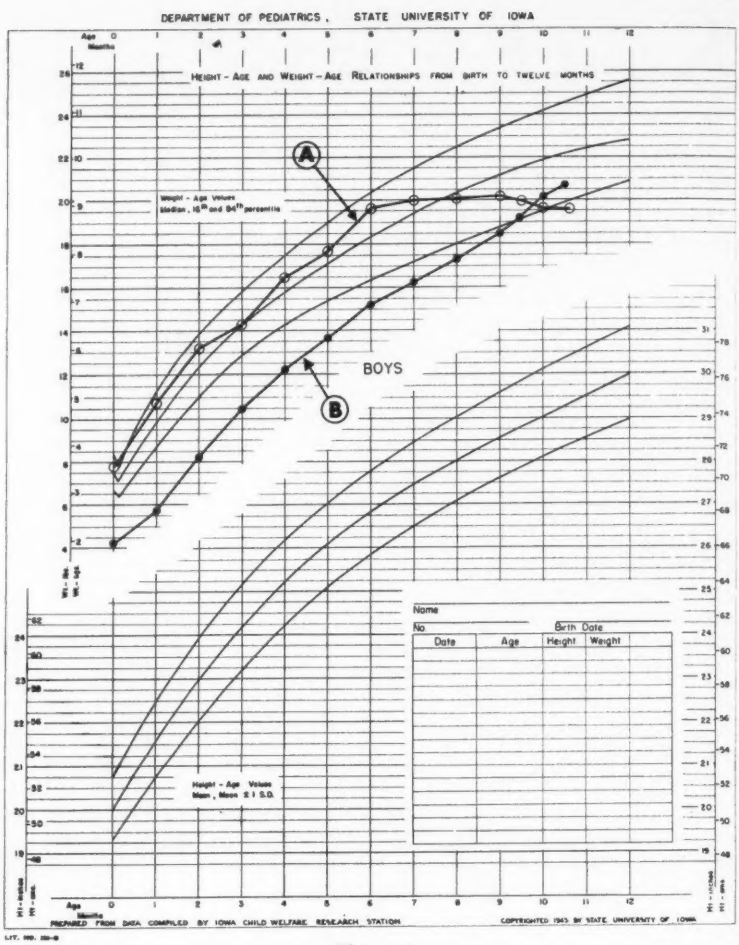

Figure 1

In addition to general body growth, it is important for the physician to be well acquainted with the various modes of growth of different organ systems. The central nervous system accomplishes fifty per cent of its postnatal growth in the first year after birth, 75 per cent by the third year, and over 90 per cent by the seventh year. Thus it is important to record head circumference measurements as an index of cerebral growth during the first few years of life, but serial measurements are not necessary routinely in later childhood because the growth of the brain is virtually complete.

Developmental behavior is equally as important as physical growth to the pediatrician. This aspect of growth and development will be covered in this Symposium.

Growth is influenced by many factors in this area. It is well known that heredity, sex, race, illegitimacy and prematurity are all important factors to be considered in assessing growth. Congenital malformation of almost any body system, malignancy, poor environmental hygiene and multiple trauma all may contribute to growth failure. Growth failure may be on a primary psychologic basis and emotional factors often contribute to growth failure by complicating the primary organic disease. In this Symposium, however, no attempt will be made to cover all facets of the problem but only certain selected aspects of physical and developmental growth.

In certain cases no demonstrable cause can be found to explain the growth failure, thus no specific treatment is possible. Unfortunately, in other cases the cause when found may not be correctable. When the

physician is satisfied that he has made a thorough examination of a child with growth failure, and that he has followed the child for a sufficiently long period of time to be certain that no etiologic factors have been overlooked, he must conclude that the child is small but otherwise healthy. His task is not finished, however, until he persuades the parents and ultimately the child to accept the fact that some human beings are small and for this there is no cure.

ENDOCRINE ASPECTS: Doctor Keele

MOST OF THE hormones have an important part in the linear growth of the young, but in contrast to widespread belief, endocrine dysfunction is not a common cause of growth retardation. Bone, nutritional, metabolic, renal and circulatory disturbances as discussed by other participants in this symposium are more common causes of growth retardation and should be considered first in the work-up of any child with this problem. However, untreated endocrine dysfunctions are associated with growth failure, and for this reason an evaluation of the linear growth becomes an important part in the diagnosis and the management of endocrine disorders.

No one hormone is solely responsible for influencing growth. Somatotrophin (growth hormone), thyroid hormone, the sex hormones, and probably insulin all participate in the growth of the child, and for this reason it is probably best to think of the hormones as being "synergestic" in their influences on growth. It is impossible at the present time to quantitate the influences of each of these on growth but that they all do influence growth has been adequately documented. This will be shown below.

There are several lines of evidence to support the role of somatotrophin in human linear growth. First, patients with organic lesions of the pituitary gland have growth *retardation;* this has been shown in patients with craniopharyngiomas,[3] Hand-Schuller-Christian Disease,[5] and possibly in toxoplasmosis.[6] However, these are usually associated with lack of other trophic hormones which may have had some casual relationship to the dwarfism. Secondly, growth hormone, extracted from the pituitary glands of man and monkeys causes linear growth in pituitary dwarfs.[1,23] Thirdly, eosinophilic adenomas of the pituitary gland cause gigantism before epiphyseal closure and acromegaly in adults.[7] Fourthly, Daughaday and co-workers[8] have shown that normal human serum contains a factor which stimulates the in vitro incorporation of sulfate-S^{35} into cartilage from hypophysectomized rats. Children and adults seem to have similar levels of this factor. The factor is almost absent from the serum of patients with pituitary dwarfism, Sheehan's syndrome (post partum necrosis to the pituitary), post-hypophysectomy lesions, and pituitary neoplasia. This factor tends to be increased in patients with active acromegaly and is normal in patients with inactive acromegaly. The sulfation factor is increased in the sera of hypopituitary patients after treatment with primate growth hormone. Although this factor may not be growth hormone, it is at least growth hormone dependent. Read[9] has obtained similar results by using an anti-human growth hormone serum produced in rabbits. That children and adults have similar levels is consistent with the observation by Gemzell and Heijenskjold[29] that growth hormone content of the pituitary glands does not change with age.

Somatotrophin probably increases human growth by protein anabolism. This has been shown to be true in animals,[10] and is associated with nitrogen retention. Somatotrophin also causes nitrogen retention in man.[1,24,12] One can speculate that somatotrophin causes human growth by accelerating chondrogenesis and bone growth just as it does in the rat.[11] There is also calcium and phosphorous retention in the treatment of pituitary dwarfs with somatotrophin.[1] Growth hormone does not cause significant bone maturation.

It is difficult to make a positive diagnosis of pituitary dwarfism in early childhood. A specific test for demonstrating growth hormone has not been available; the sulfation factor test described by Daughaday et al.[8] or the immunologic method of measuring GH

described by Read[9] may prove of value in this regard in the future. However, the diagnosis can be made with a high degree of precision if a local lesion such as a craniopharyngioma, a glioma, or Hand-Shuller-Christian Disease is demonstrated in association with dwarfism. Although such cases are rare, these lesions should be looked for by getting x-rays of the skull, especially if diabetes insipidus is present. In the majority of cases such a lesion is not present and the diagnosis is made by demonstrating the absence of the other pituitary hormones. Specific tests for the various pituitary trophic hormones are not generally available and for this reason it is necessary to look for deficiencies in the action of their target organs. Secondary hypothyroidism may or may not be present. Signs of severe hypothyroidism such as myxedema, sluggishness, constiptation are rarely seen; some may show minimal findings such as coolness and mottling of the skin and moderate lowering of the thyroid function studies. Many may have no evidence of thyroid dysfunction other than a retardation of bone age which cannot be attributed solely to thyroid hormone deficiency. Likewise, evidence of secondary hypoadrenocorticism may be absent. Occasionally one will be seen with hypoglycemic episodes and insulin sensitivity. Such findings if present are very helpful in making the diagnosis before puberty.

Of course, lack of gonadotrophic activity cannot be demonstrated until after the expected time of appearance of puberty. This can be demonstrated by the persistence of sexual infantilism and a urinary follicle stimulating hormone level of less than six m. u. per 24 hours. A positive diagnosis of pituitary dwarfism can be made if no gonadotrophic activity can be demonstrated in a dwarf with evidences of secondary hypothyroidism and hypoadrenocorticism. It is often difficult, however, to distinguish these individuals from patients with another entity called delayed puberty. In this condition the patients mature sexually and have their pubertal growth spurt two to four years late in contrast to patients with pituitary disease in which the sexual infantilism and dwarfism persists indefinitely. These patients with delayed onset of puberty also frequently give a history of lagging slightly behind their contemporaries in growth throughout childhood just as in hypopituitarism. They also tend to have a two to three year delay in bone maturation. The other parameters of pituitary function are normal. The cause is completely unknown; however, a positive family history of delayed adolescence can often be obtained, which may indicate a genetic trait.

The treatment of pituitary dwarfism presents a problem simply because there is no ideal drug commercially available. It has been well documented that primate growth hormone will result in growth of these individuals. For obvious reasons, this material is scarce, and the effectiveness of animal growth hormone in man has not been demonstrated. It is hoped that an active principle may be obtained from animal growth hormone which will be effective in man.[23]

Sex hormone treatment is indicated in proven hypopituitarism to produce a growth spurt and sexual maturity. Sometimes sex hormone treatment is indicated in delayed puberty especially when there are psychological problems relating to the sexual infantilism and delay in growth spurt. There is considerable controversy concerning the effectiveness of the androgens in children with other non-specific causes of growth failure. Sobel et al.[17] think that methyltestosterone produces more bone maturation than growth resulting in impairment of eventual stature. Bayley and co-workers[16] in testing its use in immature boys think that treatment with small doses, 10 mgm per day, produces more ultimate growth than occurs in untreated patients. They think that this dosage has the advantage of avoiding undesirable masculinization. We must leave this question open until further clinical trials have proven its effectiveness, or the effectiveness of some closely related compound.

That thyroid hormone influences linear growth is shown by the marked dwarfism in cretins and the cessation of growth in acquired hypothyroidism. Normal growth can be attained when these patients are treated with physiologic dosages of thyroid hormone. Thyroid hormone is moderate in its

bone growth promoting activity, but is very active in promoting bone maturation.[11] In the child, absence of thyroid hormone results in marked lack of growth of bone, retention of infantile skeletal proportions, and marked retardation of bone age.

The diagnosis of cretinism is difficult to make in the neonatal period. Most of these patients appear normal at birth but by three weeks of age the first symptoms usually appear and include: constipation; wrinkling of the brow; poor feeding; and cool, mottled extremities. The typical picture of cretinism with the characteristic facies of myxedema does not appear until three to six months of age at which time the chances of permanent mental damage are increased. In the absence of thyroid hormone the brain fails to grow in the newborn rat[14] and histologic damage has been demonstrated in man.[21] For this reason it is important to make the diagnosis as early as possible. In any neonate with the above symptoms retardation of bone age and lowering of the Butanol Extractable Iodine and Protein Bound Iodine should be looked for. Treatment with thyroid hormone should be started if cretinism is suspected in order to allow maximum brain growth and preserve potential mental development.[22] Various regimens are available in the literature.[7,15] Normal serum levels for BEI and PBI are also available.[15]

Children with acquired hypothyroidism usually are not so dwarfed and some may be in the normal range for height-age. These patients usually do not have permanent mental damage because the brain has finished most of its growth at the time of occurrence of the disease. A diagnosis ordinarily can be made by finding a lowered PBI, BEI, and radioactive iodine uptake. Normal mental activity and linear growth can be expected on treatment with physiologic dosages of thyroid hormone.

Untreated diabetes mellitus will result in growth retardation; rarely, however, does juvenile diabetes escape detection and treatment before marked growth failure occurs. Poorly controlled diabetes will result in dwarfism.[2,20] This seems to be related to hypersensitiveness to quick acting insulin and has not been commonly seen since the advent of the long-acting insulins. The mechanism by which insulin increases linear growth is not known. Animal experiments suggest that insulin is essential for protein-anabolism and that it is essential for the maximum anabolic response to growth hormone.[18]

Dwarfism occurs ultimately in sexual precocity. Sexual precocity is associated with excess sex hormone production. This occurs in constitutional precocious puberty, androgen producing lesions of the adrenal, and in certain gonadal tumors. Excesses of sex hormones, androgen and estrogen, in early childhood produce increased growth; however, the rate of bone maturation is increased over linear growth resulting in early closure of the epiphyses and ultimate sacrifice of adult height. Treatment to prevent the dwarfism of constitutional precocious puberty is not available. In congenital adrenal hyperplasia, early rapid growth and early fusion of epiphyses can be prevented by treating the patients with daily physiologic dosages of the glucocorticoids which inhibit the excess androgen production by the hyperplastic gland. In adrenal and gonadal tumors, surgical removal is desirable.

Dwarfism also occurs in gonadal aplasia in females, a syndrome first described by H. H. Turner,[25] and later by Varney et al.,[26] Albright et al.[27] and Wilkins.[28] These patients have, in addition to dwarfism, elevated urinary levels of gonadotrophins, sexual infantilism, and other congenital defects. About 80 per cent are sex chromotin negative (male pattern). The cause of the dwarfism is unknown. Femininization and some growth can be produced by estrogen treatment.

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METABOLIC ASPECTS: Doctor Kirkman

ONE DEFINITION of metabolism is: "the sum of all the physical and chemical processes by which living, organized substance is produced and maintained."[1] With this broad scope in mind, we shall cite not only those disorders conventionally accepted as metabolic in nature, but also those disorders not covered by speakers on other categories.

When confronted with an actual growth failure of possible metabolic origin it may be diagnostically helpful for the examining physician to think in the same sequence as occurs in the utilization of food and conversion of food to energy or tissue—from grocery store to glycogen stores.

The relative abundancy of food and the means for its purchase in this country make starvation or malnutrition an unusual—but not unseen—occurrence. In addition to economic deprivation, inadequate intake of nourishment from other causes must be considered in instances of growth failure. The question of whether a new, breast-fed infant is getting enough milk is a common one. The new mother may misunderstand instructions for formula or may fail to learn that the holes of rubber nipples may have to be widened. Over-conservatism or failure to appreciate the greater individual caloric needs of a more active infant may result in undue growth failure in an older premature infant in the nursery. A monotonous diet is as discouraging to children as to adults. Specific dietary deficiencies, such as vitamin or amino-acid deficiency are rare and overly emphasized in this country. The lay recognition of the nutritional need for milk in infancy and childhood has obviated most of the deficiencies except those of vitamin C and iron, items in which milk is deficient. Even these two deficiencies can occur with surprisingly little departure from expected weight. However, the possibility of a specific deficiency must be considered when abnormal diets are imposed on a child for reasons of allergy or absorptive problems. While the practicing physician may prescribe various vitamins in the hope of placating the parents or perhaps actually increasing the appetite of the child, he will generally be disappointed in this regard and may, in the specific case of idiopathic hypercalcemia or in the general case of detracting from the pursuit of the true diagnosis, do more harm than good.

Failure to feed due to neuromuscular, psychologic, or mental deficiencies will be discussed by others on this panel. Conditions which interfere with delivery of food to the intestines may result in growth failure and should be apparent from a history of vomiting or difficulty in feeding. These conditions include posterior-nasal atresia, rumination, esophageal anomalies, muscular disorders of gastric rhythm, pyloric stenosis, duodenal atresia, etc. A deficiency of pancreatic enzymes, as in fibrocystic disease,[2] may account for the poor digestion of an otherwise

adequate diet. Celiac disease[3] in children and older infants, and steatorrhea in the younger infants are expressions of an inadequate absorption of digested food-stuff by the intestinal wall. Both disorders are characterized by loose stools and increased concentration of unabsorbed fat in the stools. In celiac disease the oral administration of glucose or vitamin A produces less elevation of the blood-levels of these substances than in normal persons, yet the intravenous glucose tolerance curves in celiac disease may be normal.[3] These tests illustrate the defective intestinal absorption that exists in celiac disease. The various other forms of diarrhea may also cause failure in growth because of poor absorption.

Once absorbed, further actions on the intermediates may be impaired because of hepatic injury—either metabolic, as in the cirrhosis of galactosemia,[4,5] fibrocystic disease, and one of the glycogen storage diseases—or infectious. In the Von Gierke's type of glycogen storage disease, the liver lacks glucose-6-phosphatase and miserly refuses to return to the general circulation, in times of need, that glucose which it has received in times of feeding.[6] The liver enlarges from this accumulation of glucose as glycogen in Von Gierke's disease, and the administration of epinephrine fails to produce the usual elevation of blood glucose.

Growth may be impaired due to specific abnormalities of the chondro-osseous framework, as chondrodystrophy, Morquio's disease, and osteogenesis imperfecta. In this regard it is wise, of course, to search for those constellations of signs which portray the condition as a recognizable syndrome or disorder—as the characteristic disproportions of the chondrodystrophic dwarf, or the features of gargoylism.[7] In the latter instance, and in cases of renal disease, idiopathic hypercalcemia, lipidoses,[8] and untreated galactosemia, the chemical manifestations are becoming known, but the exact ways in which synthesis of tissue is impaired are not known. But for the examining physician, the problem of diagnosis of metabolic growth failure lies not among such physically apparent disorders as chondrodystrophy, Morquio's, celiac, glycogen storage, Niemann-Pick disease, etc; the relatively striking physical findings in these instances will cause the examiner to make the diagnosis or consult the literature pertinent to the appropriate physical signs. Indeed, some of these disorders are not primarily of concern as growth problems. The concern is rather with the disorders with the more elusive or misleading findings. Notoriously belonging to this group are fibrocystic disease,[2], idiopathic hypercalcemia,[9] galactosemia,[4,5] and the renal tubular defects.[10] It behooves us, therefore, to become familiar with the suggestive symptoms of these disorders and the laboratory tests appropriate to their diagnosis.

The victims of fibrocystic disease, synonymously known as cystic fibrosis of the pancreas or mucoviscidosis secrete a mucin of high viscosity and a sweat of high concentration in electrolytes. As a consequence of the abnormal mucin, obstructions of the pancreas and of the broncial and biliary trees may occur. Meconium ileus, impaired digestion of proteins, and chronic respiratory distress or infection may result. The diagnosis of fibrocystic disease is confirmed by the finding of a deficiency of duodenal trypsin or an excessive concentration of chloride in sweat. Children with the milder forms of idiopathic hypercalcemia may exhibit only anorexia, irritability, and some vomiting. Listlessness and obvious growth failure follow. Elevation of serum calcium and moderate albuminuria occur. Young infants with untreated galactosemia have hepatomegaly, vomiting, severe growth failure, and often jaundice. If they do not die, they lose their jaundice but often develop cirrhosis, cataracts, and mental deficiency. The excretion of urinary substance (galactose) which reduces Benedict's solution but fails to reduce glucose oxidose test paper, is characteristic of the galactosemic child taking milk. Highly reliable enzymic blood tests exist for the diagnosis of galactosemia. Victims of the renal tubular defects may have rickets or merely non-specific symptoms accompanying growth failure. Their diagnosis is suspected by the findings on routine urinalysis, combined with determinations of serum electrolytes and bicarbonate. Urinary amino-acid determinations may be confirmatory.

Recent, very powerful tools in biochemical

investigations have clarified some of these metabolic problems and promise to unveil new causes for growth failure. But, in spite of these technical tools the fact remains that all of these presently-known metabolic disorders are tentatively diagnosable by a well-taken history and physical examination and by a selected group of more or less routine laboratory tests. From this tentative evidence the more specific or confirmatory tests may be chosen.

These metabolic diseases are more than just biochemical oddities of interest only to a small group of academic investigators. Diagnosis of these diseases provides very definite information for prognostic and genetic counseling. Early detection and treatment of such disorders as celiac disease and galactosemia may be life-saving and may permit the individual to lead a normal life. Appropriate treatment can permit the victims of fibrocystic disease, glycogen storage disease, and renal tubular disorders to lead longer and more comfortable lives. These measures can be taken with a clear conscience toward possible pollution of the human genetic stock. Most of these disorders are relatively uncommon, recessive diseases. This means that symptomless carriers of the disease, rather than the affected individual, are responsible for the perpetuation of the mutations. Indeed, by his recognition of the disorder and genetic counseling to the parents, the practicing physician has contributed his bit toward the genetic hygiene of his species.

In conclusion, metabolic disturbances often express themselves in infancy and childhood as growth failure. To the practitioner, this is both a helpful diagnostic sign and an alarm as to urgency of needed action, for metabolic causes of this failure may, if untreated, deprive children of their rightful legacy of life and growth.

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NEUROLOGIC ASPECTS: Doctor Waites

AN UNDETERMINED NUMBER of infants who do not thrive have cerebral dysgenesis (or agenesis) as the basic pathological process in their difficulty. This may be manifest in a variety of clinical forms. Dysgenesis implies that the nerve cells of the brain do not develop as normal neuroblasts, or that defective maturation interposed between the neuroblast stage and the mature neuron. Either abnormal neuroblasts or defective maturation would subsequently result in neuronal degeneration and the clinical manifestations of cerebral dysgenesis. It naturally follows that the white matter of the nervous system will not be normal if the grey matter with its nerve processes does not progress through the usual embryological stages to maturity. These conditions have been adequately demonstrated pathologically from the most severe microcephaly, with no differentiation between grey and white matter, to the most discrete nerve involvement of a single functional system of the brain.

For this discussion, we will consider the conditions that have been termed historically "cerebral spastic paralysis," "Little's disease," "double infantile spastic hemiplegia," "spastic diplegia," "spastic paraplegia," and now have the glorious misnomer of "cerebral palsy."

Little published his famous monograph in 1861 and first called attention to "the influence of abnormal parturition, difficult labor, premature birth, and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities." His clinical descriptions, observations, and deductions therefrom are legendary and have hardly been improved upon to the present. He did not believe gross injury to the brain to be the causative factor, and he did not believe meningeal hemorrhage to be a factor. He did establish the fact that the condition is often associat-

ed with abnormalities of birth, but he also states that many infants born of difficult labor and delivery would subsequently be normal. It was his opinion that the pathology was a widely spread fine lesion of the cerebral substance, and that it may affect one part of the brain more than another. He reasoned that the most common causal factor was asphyxia.

In 1885, Sara McNutt presented a thesis to the American Neurological Association concerning this same subject. This particular thesis is of much significance to us today. The clinical descriptions and pathological findings in this paper are excellent, but the erroneous deductions from these findings have remained with us to this day. She reasoned that the entire process of double infantile spastic hemiplegia was produced by meningeal hemorrhage in her cases, and consequently, hemorrhage was the etiological factor in infantile spastic states dating from birth and associated with difficult labor. This erroneous deduction has perpetuated the most amazing and unbelievable influence upon medical thought concerning spastic and related states, and only recently have other concepts gained general medical credence. Even today it is not uncommon to hear that an infant has difficulty because of "birth trauma and/or hemorrhage," whereas neither of these morbid conditions can honestly be incriminated.

In 1897, Freud summarized some of his observations in the monograph "Infantile Cerebral Paralysis." He included a critical analysis of all previous papers on this subject. This is still the most complete essay on the subject. He was the first to use the term "cerebral diplegia" in reference to bilateral symmetrical defects in the brain, not related to gross lesions. The result of gross lesions he termed hemiplegia and double hemiplegia. He proved that diplegia had its pathological origin long before birth in intra-uterine existence, and largely disproved that meningeal hemorrhage was any factor in diplegia or hemiplegia. Freud summarizes: "Premature, precipitate and difficult birth, and asphyxia neonatorum are not causal factors in the production of diplegia; they are only associated symptoms of deeper lying influences which have dominated the development of the foetus or the organisms of the mother." He was of the opinion that the cerebral paralysis results from difficult births when the brain is lacerated or contused, and that this is manifest as a monoplegia or hemiplegia, very rarely as a double hemiplegia. The anatomical changes in the brain would then be entirely different from those of diplegia. After disproving the traumatic delivery theory as a causative factor, Freud connected the prenatally developed diplegia with those cases in which the onset may occur at any age from birth even to puberty. He argued strongly for a primary neuronal degeneration of the central nervous system which might become manifest during any phase of fetal life or childhood.

In 1909, Anglane and Jacquin reported the findings in a patient with spastic diplegia in which there was neuronal degeneration in the cerebellum, as well as extensive neuronal degeneration in the cerebrum. This may be the first report of such involvement in the cerebellum. Batten, an English neurologist with more than usual interest in pediatric neurology, postulated that the cerebellum could be involved in some types of spastic disease. He believed that the cerebellum was small because of neuronal involvement, and not because of lack of the cerebro-ponto-cerebellar fibres as had been postulated by some of his contemporaries.

Collier, in 1924, again suggested that "agenesia" is a primary neuronal defect, and is responsible for essentially all cases of diplegia. His article in *Brain*, 1924, is well worth one's attention and is the primary source for these remarks.

Recent workers from the Continent have suggested that venous congestion may be an important factor in spastic states, and related conditions. Their attention has been focused on the area of the great vein of Galen and its tributaries—much investigation remains to be completed before their final answer will be forthcoming.

We may someday realize the importance of the embryology of the specific functional systems of the brain and the temporal relationship of one system to the other. The problem then resolves itself to determining the etiology of neuronal involvement in each

system that may show pathological development, and the subsequent clinical manifestations.

From the work that has been done in this field to the present time, it is safe to assume that most of the infants that fail to thrive because of central nervous system involvement had this difficulty before they were born. The clinical picture is a result of the prenatal defect. Birth trauma, hemorrhage, asphyxia, anoxia, and other such morbid factors may well contribute to the difficulty, but are not the etiology.

When the patho-physiology of cerebral dysgenesis is completely understood, it is likely that the pathogenesis of the more subtle neurological disturbances in infancy will be quite clear.

PSYCHOLOGICAL, SENSORY and PERCEPTUAL ASPECTS: Doctor Richardson

COMPARATIVELY LITTLE is known about normal psychologic, sensory and perceptual growth and development, especially in infancy. Our increasing awareness of the pathology in these areas, however, is stimulating quite a bit of research, the results of which promise to be rewarding.

The earliest sign of psychologic, sensory or perceptual dysfunction is most frequently seen in the delayed or faulty acquisition of speech or language, the latter being that symbolic function which includes all forms of communication.

Best and Taylor describe normal speech development (including the symbolic function of language): "The first stage in the development of speech is the association of certain sounds (words) with visual, tactile and other sensations aroused by objects in the external world. These associations are 'stored' as memories. After definite meanings are attached to certain words, pathways between the auditory area of the cortex and the motor area for the muscles of articulation become established, and the child attempts to formulate and to pronounce the words which he has heard. This act of verbal expression involves the coordinated movements of a large group of respiratory, laryngeal, lingual, pharyngeal and labial muscles. Later, as the child is taught to read, auditory speech is associated with the visual symbols of speech and, finally, through an association between these and the motor area for the hand, the child learns to express his auditory and visual impressions by the written word." It has been stated truly that speech is the greatest interest and the most distinctive achievement of man!

Language development has both physical and psychological components. It requires adequate integrity of the peripheral nervous system (sensory capacities), of the central nervous system, and adequate integrity psychologically. Anatomical integrity of the articulary apparatus is necessary of course.

We might review briefly the normal sequence of speech development. The child begins to use single words to express his needs usually between ten months and two and one-half years of age. Two or three word phrases are introduced between eighteen months and three and one-half years, and an ever increasing vocabulary follows. Although the period of speech development may normally extend from nine months to three and one-half years, the period between one and two years of age is in most children that of maximum rate of speech development, and after this period speech is acquired with less facility.

It has always been recognized that retarded mental development is closely allied with delay in the utilization of speech skills as well as in decreased language content. Mental retardation interferes with full comprehension, although all but the most severe who have normal hearing understand simple speech associated with the basic needs of life.

If hearing is impaired, the child may fail completely to speak, or language development may be retarded and articulation defective. The intelligent hard of hearing child may show an interest in oral communication and may attempt to compensate for his hearing disability through lip reading. If the hearing loss is severe and the child is able to hear only noises he may be certified erroneously as mentally defective. General response to sound in such a child might be regarded as an indication of normal hear-

ing, and failure to understand fully what is said, or to make suitable progress in school, might be taken as a sign of retardation. Inasmuch as speech is learned chiefly through the hearing mechanism by imitation, the child learns to speak not only *because* he hears, but *as* he hears.

If development of speech is delayed well beyond the range of normal, and the child's ability to formulate his thoughts into language is grossly inadequate for his age, the condition may be described as a developmental dysphasia or a central communication disorder. Such a disorder may be associated with spastic and related conditions, or may exist in the absence of any obvious underlying neurologic disorder. It may be the result of some cerebral dysgenesis producing delay in the use of the symbols of speech. The child with delayed neurologic maturation of the central processes involved in the use of expressive speech may have adequate understanding of language. He may be friendly and cooperative in his attempts to communicate with others and yet be unable to express himself through speech. He may appear to have little or no interest in the imitation of sounds, having been a silent child who made little attempt to vocalize or to babble during infancy. He may show signs of frustration when he cannot make known his meaning and may rely chiefly on gesture or well-developed mime to achieve his essential need for communication. This has been called expressive or motor dysphasia.

In other instances there may be lacking or limited appreciation for the meaning of spoken language which is not related either to peripheral auditory impairment or subnormal intelligence. This condition has been described as receptive dysphasia or congenital auditory imperception. These children may also use gesture to supplement their limited means of verbal expression. They generally show little interest in speech as a means of communication, often fail to attempt speech, and make little or no attempt to use the visual sensory pathways such as lip reading. The child who has no understanding of language and who cannot express himself orally is said to have a mixed type of dysphasia. It is actually doubtful that there is a pure form of dysphasia at all.

There are as yet relatively few studies on perceptual deviations as such. However, the studies which have been done on children with learning disabilities such as specific reading disability, or dyslexia, have led some of us to believe that perceptual disorders are not uncommon in children with delayed language development. Though intelligence depends on the functioning of many parts of the brain, certain areas have been located that investigators believe are more particularly associated with perceptive processes. Association paths between these areas are known to exist, and there are always nerves which relay impressions from the sense organs to the brain centers, or from the brain to motor and sensory mechanisms. Many combinations of injury or faulty development are therefore possible, and the potential effects on the child's perceptive and thought processes are legion. Also a sensory defect interferes with perception in the range of action of the defective sense. Therefore, if there are also sensory handicaps, their exact nature and their effect on perception need to be carefully determined. It must also be pointed out that perceptive or thought defects can be caused by emotional problems or personality distortion.

Strauss defines perception as intermediate between sensation and thought, calling it "the mental process which gives particular meaning and significance to a given sensation and therefore acts as the preliminary to thinking. It is the means by which the individual organizes and comes to understand the phenomena which constantly impinge upon him." He describes two particular aspects of perception as follows: (1) Perception, in terms of Gestalt psychology, is a psychologic process in which each part of a whole is seen or heard or felt in relationship to the other parts, resulting in a figure which is recognized immediately and uniquely. (2) Another characteristic of normal perception is the fact that a whole is perceived as a foreground figure against a background. We have seen how some children with central nervous system dysfunction or dysgenesis are unable to coordinate parts of a simple whole, like the sides of a triangle,

into a correctly integrated form. In addition, when figures of test material are presented against a background, these children cannot differentiate the foreground from the background. Moreover, in trying to assemble test material which involves the use of motor, visual, or hearing abilities, some children may be incoherent and disorganized in their attempts. Many now feel that children with specific reading disability or other communication disorders have this type of visual-motor perceptual difficulty. Perceptual deviations have been commonly attributed to specific neural lesions, but they seem similar to performance patterns which are normally expected of children at lower levels of perceptual maturation; therefore, the possibility of retarded or arrested maturation should be considered in evaluating what are apparently perceptual defects.

Interference with the development of speech can occur if emotional growth is not progressing harmoniously. There may be little need or desire for contact or communication with others. Speech, once acquired, may not be used freely or may be almost entirely inhibited. Extreme shyness or self-consciousness can hinder the gradual development and stabilization of speech. If the arrested or defective development of speech is the result of, or associated with, true psychogenic retardation, there will probably be signs of emotional difficulties and maladjustment in other respects. It is not uncommon, however, for the emotional disturbance to be secondary to the speech disorder, especially where much frustration has been experienced by a child with developmental dysphasia or unintelligible articulation. Many children refuse to speak and take refuge in silence when they realize their inability to communicate effectively, or when they are subjected to repeated and misdirected correction of their speech at home.

There are many so-called functional or environmental causes for delayed speech. These would include prolonged or serious illness during the first year of life which interferes with the baby's vocal play and prelanguage activity; over-protective or rejecting parents; poor speech models in the home; the lack of a mother or a mother-figure in close relationship with the child; over-anxious parents who try to force or cajole the child into speaking; etc.

The delayed establishment of cerebral dominance used to be considered an etiological factor in stuttering. This has not been borne out, but there is strong evidence that delayed development of cerebral dominance may be causal in delayed speech. Dominance develops gradually and is normally established between the age of three and six years. Nielsen points out that many children show definite laterality at one year. Speech readiness usually develops along with dominance and if the latter is delayed, the former is usually similarly retarded. Evidence has seemed convincing that there is a higher incidence of speech disorders, learning disabilities, and behavior disorders in children whose dominance is not "set," that is, who have a lack of unilateral hand preference, than in those who are strongly right or left handed. However, there is lack of agreement as to whether a cause and effect relationship exists or whether these conditions simply occur concurrently. Cerebral dominance is said to involve not only hand dominance but also eye dominance. Berner and Berner concluded from the study of 500 cases with and without reading difficulties, defects of speech, and allied visual-motor disorganization, that unless the controlling eye in binocular vision is on the side of the dominant hand, a chain of symptoms referable to eye-hand confusion is likely to occur. Further study is needed to throw light on these controversial issues and help to increase understanding of what disturbed dominance means.

In summary, the delayed or faulty acquisition of speech or language may be one of the earliest and most signifcant signs of psychological, sensory or perceptual dysfunction. Faced with such a child, we have to decide whether this is due to mental deficiency, severe or partial hearing loss, to developmental dysphasia or to psychogenic factors. There are numerous environmental factors which may also be etiologic. The relationship between cerebral dominance and communication skills has yet to be clarified.

CARDIAC ASPECTS: Doctor Cayler

THERE IS an abundance of clinical information concerning growth failure or retardation in children with congenital heart disease, but the basic mechanisms of growth failure in these patients is not well understood. Everyone working in the field has opinions based on clinical observations. There are several reports of clinical studies documenting abnormal growth patterns and changes in growth patterns after surgical therapy. There is, however, very little information concerning the actual etiology of growth failure in these patients.

One bottle-neck to etiologic investigations has been the lack of information concerning normal hemodynamics in infants and children. Without normal data it is impossible to intelligently interpret the significance of abnormal data from patients with heart defects. The reasons for the pausity of normal data is due to the fact that no easy clinical method is available for obtaining detailed accurate hemodynamic information. At present the cardio-pulmonary laboratory is the only place where these type studies can be performed. Investigators are justifiably hesitant about subjecting normal infants to the type studies needed. Another source of difficulty is in actually obtaining enough voluntary, or involuntary in the case of infants, cooperation to allow controlled hemodynamic measurements.

The circulatory system of course is vital to all the body functions including growth. It normally carries oxygen and nutrients to the capillaries and maintains an adequate diffusion gradient to allow exchange of gases and metabolites between the vascular system and the tissues. Congenital heart disease is generally considered to interfere with normal tissue metabolism and growth by resulting in:

1) a decrease in systemic arterial oxygen saturation (right to left shunt) or 2) a decrease in systemic cardiac output. Although in general the degree of growth retardation correlates with the severity of the heart defect, exceptions are not uncommon both in terms of marked growth failure with minimal heart disease and minimal or no growth failure with very severe heart disease.

Growth failure due to systemic arterial unsaturation and decreased tissue oxygen delivery is easy to appreciate. When severe, even the compensatory mechanisms of polycythemia, changes in the hemoglobin-oxygen dissociation curve and perhaps increased cardiac output do not allow adequate basal or resting oxygen delivery. Some observers have reported an apparent compensatory decrease in oxygen utilization or a decreased basal metabolic rate in these patients,[1] but this has not been found by others.[2] Growth failure may not be a good term for patients with cyanotic heart disease. Growth retardation is more appropriate since the majority of these patients eventually attain full physical development.[3] It is of interest that the growth of patients with cyanotic heart disease is occasionally actually greater than normal. This is seen most commonly in patients with isolated pulmonary stenosis. It is possible that the explanation for this is related to the increased caloric intake and limited energy expenditure (e.g. marked physical inactivity) which is often seen in these patients.

It is also easy to understand how an actual decrease in basal or resting cardiac output may interfere with tissue metabolism and growth. Such a decrease has been demonstrated in adults with congenital heart disease but has not been found in children.[4] Until recently no output studies had been performed on infants with congenital heart disease. Table one lists the data on systemic cardiac output for a group of patients with congenital heart disease whose ages range from ten weeks to forty years.[2] The difference in resting cardiac output between the older groups is apparent. No difference in resting cardiac output was demonstrated however between the control (class 1) or diseased (classes 2, 3, 4) infants or smaller children, even though growth retardation in these later patients was usually quite marked. It is possible that the difference between the cardiac output necessary to sustain life and the cardiac output necessary to permit normal physical development is so small in infants that our relatively crude methods of measurement are inadequate to demonstrate the difference.

RESTING CARDIAC INDEX				
	B.S.A. (m^2)	AGE	SEVERITY OF HEART LESION	CARDIAC INDEX (L/Min)
1.	0.2-0.5	<2 yrs.	1. Class 1	—
			2. Class 2	5.0
			3. Class 3 & 4	5.0
2.	0.5-1.0	5-10	1. Class 1	5.0
			2. Class 2	5.0
			3. Class 3 & 4	5.0
3.	1.0-1.5	10-15	1. Class 1	5.0
			2. Class 2	4.0
			3. Class 3 & 4	3.0
4.	1.5 +	15+	1. Class 1	4.5
			2. Class 2	3.5
			3. Class 3 & 4	3.0

TABLE ONE

The effect of congenital heart disease on resting systemic cardiac output in infants and children.

The pattern of growth failure due to congenital heart disease is one of greater interference with weight growth than length or height growth.[5] This is best demonstrated on an anthropometric chart where one almost always observes that the weight percentile is significantly lower than the length or height percentile in these patients. If this pattern of growth failure is not seen it is most likely that the growth failure is not due to heart disease.

Physical growth failure due to congenital heart disease is not static. Growth retardation is usually not progressively more severe after infancy. Although growth usually continues at about the same rate after infancy, appreciable spontaneous improved growth is not infrequently seen. In a series of infants and children with ventricular septal defects, nine of thirty-two (28 per cent) showed improved growth after the age of one year.[6] Engle[7] and others[8,9] have shown that surgical correction of patent ductus arteriosus in children is often not accompanied by improved growth. Present data suggests that the earlier the surgical correction is performed in relation to the onset of growth failure, the greater is the chance of obtaining normal growth.

Although puberty may be delayed in the child with severe congenital heart disease, there is no good evidence to indicate that the heart disease results in significant secondary endocrine abnormalities which may cause growth failure. Rarely a child may have both congenital heart disease and an endocrine deficiency. Correction of the heart disease does not cure the endocrine problem.

In conclusion, it is well to emphasize that there are often primary or associated non cardiac reasons for growth failure in children with congenital heart disease. These include familial (or hereditary), emotional, nutritional, metabolic, and infectious conditions.

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INFECTIOUS ASPECTS: Doctor Riley

CHRONIC or repeated infection is probably the most frequent cause of malnutrition and growth failure in American children and a careful search for infection is indicated in every child with growth failure. As a rule, infection usually produces a decrease in appetite and interferes with proper digestion and absorption of food stuffs. Fever and other influences of infection often increase metabolic need for nutrients. Furthermore, depression of the bone marrow with resulting anemia and oxygen lack to tissue may occur as a result of infection. The inactivity which often accompanies illness may create negative nitrogen and calcium balances in a child and thereby interfere with growth. The effect of infection on growth may be either local or general. Apart from the obvious effects of anorexia and fever, however, the deceleration or arrest in growth that occurs with infection is unexplained. Gardiner-Hill[1] states that certain infections may cause a premature "senescence of growing cartilage" with calcifica-

tion. In the case of acute infection, there appears to be a compensatory mechanism which permits growth to resume its intrinsic pattern during the recovery phase. However, if infections are chronic or recurrent, even though relatively minor, growth may be significantly affected.

The most extreme examples of growth failure due to infection are seen in regions where parasitic diseases such as malaria, hookworm and tapeworm infestations occur. It has been shown that experimental animals that are rendered anemic and hypoproteinemic will utilize dietary protein preferentially for the formation of hemoglobin rather than for reconstruction of body mass and presumably skeletal development.

A study was carried out by the Social Medicine Unit of Oxford University concerning the effect of common childhood diseases on skeletal development. This study involved 650 individual children during more than 2000 child years of observation. These children experienced measles, chicken pox, scarlet fever, pertussis, respiratory, skin and intestinal infections as well as other common childhood diseases. The final statistical evaluation revealed that there was a stunting effect when the heights of a group of children who had been ill in any given time were compared with a similar group who had remained healthy.[2]

Chronic or recurrent infection of virtually any system may result in growth retardation.

Respiratory Infection:

It is well known that children with recurrent respiratory infections such as acute tonsillitis and otitis media may exhibit growth failure if there is a frequent occurrence of these infections even though the individual infection may not be clinically severe. The failure in growth of children with chronic pulmonary infections including bronchiectasis, cystic fibrosis of the pancreas, tuberculosis and pulmonary mycoses is well documented. Children with bronchial asthma are prone to develop pulmonary infections and may exhibit growth failure on this basis.

Urinary Tract Infection:

Pyelonephritis and congenital anomalies of the urinary tract should be searched for in any child who has growth failure unexplained by other causes. West and Smith[3] investigated the effects of renal insufficiency on growth. Virtually all of their patients who showed marked disturbance of growth had either bilateral involvement such as renal hypoplasia or bilateral obstructive phenomena with superimposed infection. The vast majority of patients with growth failure of renal etiology exhibit chronic renal acidosis. It is likely that the decreased food intake, due to the need to ingest large quantities of water leaving little time or gastric space available for food intake, may explain in part the resulting growth failure in such patients.

Gastrointestinal Infections:

It is also well known that infants and children with chronic or recurrent bacillary dysentery or parasitic infestations of the intestinal tract will exhibit growth failure. The constant loss of minerals and other nutrients in the stools of these patients may contribute to the growth retardation.

Infections of the Osseous System:

While local infection can increase bone length by metaphyseal revascularization, chronic osteomyelitis usually results in generalized growth retardation. Destructive vertebral lesions with secondary collapse and wedging can reduce height.

There are of course many other types of chronic or recurrent infection that could be mentioned. It is safe to say that chronic or recurrent infection in any system can result in growth retardation in certain children. It is also important that certain fundamental defects such as agammaglobulinemia which may predispose to infection in any one or several body systems not be overlooked.

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Defects in the chest wall permitting herniation of the lung may arise from a variety of causes. Good repair can easily be obtained using local chest wall tissue.

THORACIC LUNG HERNIA

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LUNG HERNIA may be defined as a condition in which a portion of the lung protrudes beyond the normal confines of the thoracic cavity. The herniated lung may or may not be contained in a sac of parietal pleura and is characteristically reduced at rest and herniated only on coughing, straining or expiration against the closed glottis.

Such hernias may be classified as thoracic, cervical, mediastinal or diaphragmatic according to their location. Etiologically they may be congenital or acquired, the latter being traumatic (accidental or surgical), spontaneous or pathologic.

A case of acquired, traumatic thoracic lung hernia, associated with underlying bronchiectasis, chronic lung abscess and broncho-cutaneous fistula, is presented.

Case Report

R. O. McD., a 31 year old white male construction worker, first became ill with chills, fever and productive cough in early February 1958. He was hospitalized first in his community hospital and then transferred to the Veterans Administration Hospital, Amarillo, Texas, in early March 1958. Chest roentgenograms revealed a large cavitary lesion in the posterior portion of the left lower lobe. A course of antibiotics was given with some improvement.

From the Department of Surgery of the University of Oklahoma Medical Center and the Thoracic Surgical Service of the Veterans Administration Hospital, Oklahoma City, Oklahoma.

Figure 1. Pre-operative view of patient showing the left posterior chest wall defect during normal respiration.

On March 12, 1958, he was transferred to the Veterans Administration Hospital, Oklahoma City, with the diagnosis of lung abscess of the left lower lobe and empyema. A left thoracentesis was performed shortly after admission, and 160 cc. of thick green pus was obtained which on culture produced *Klebsiella pneumonia.* He was given chloroamphenicol, and on March 17, 1958 a closed thoracotomy (tube) drainage was carried out on the left. On April 9, 1958 a large pleural air leak was noted indicating a bronchopleural fistula. On April 17, 1958 open thoracotomy drainage was performed with resection of approximately 8 cm. of the eighth and ninth left ribs posteriorly. A thick-walled empyema cavity was encountered which communicated with an abscess in the superior segment of the left lower lobe. The cavity was packed, and resolution of the purulent infection occurred. The patient was discharged from the hospital on May 5, 1958.

He did not return until his readmission on September 18, 1959, seventeen months later. He stated that he had been relatively well in the interval, but that he was troubled with the chest wall defect through which his lung bulged on coughing and which required a dressing because of the slight to moderate constant drainage. He denied cough, sputum, fever, pain or dyspnea.

Physical examination revealed a healthy appearing adult male in no distress and with normal vital signs. No abnormalities were noted except for a defect 6 cm. in diameter in the left posterior chest wall (figure 1). On coughing lung tissue with a rather glistening surface would herniate through the defect with the escape of a small amount of air (figure 2). There was no evidence of local purulent infection, and the lungs were otherwise normal to examination.

A complete blood count and urinalysis were normal. Cultures of the chest wall lesion grew *Proteus mirabilis.* Standard chest roentgenograms showed no abnormality of the lung fields except for the rib defects on the left. Bronshography on September 24, 1959 revealed contraction of the left lower lobe basilar bronchi with spillage of the contrast medium into the defect via the bronchi of the superior segment of the left lower lobe (figure 4). The remainder of the bronchial tree was normal.

On September 28, 1959 left thoracotomy was performed. The remainder of the eighth

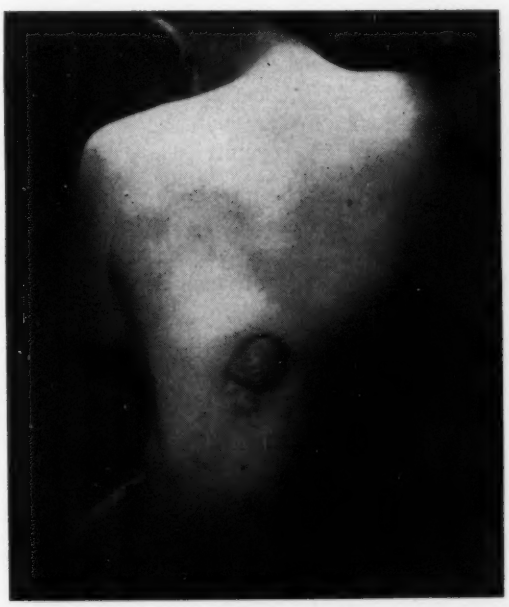

Figure 2. Pre-operative view of patient showing lung herniating through the chest wall defect on coughing.

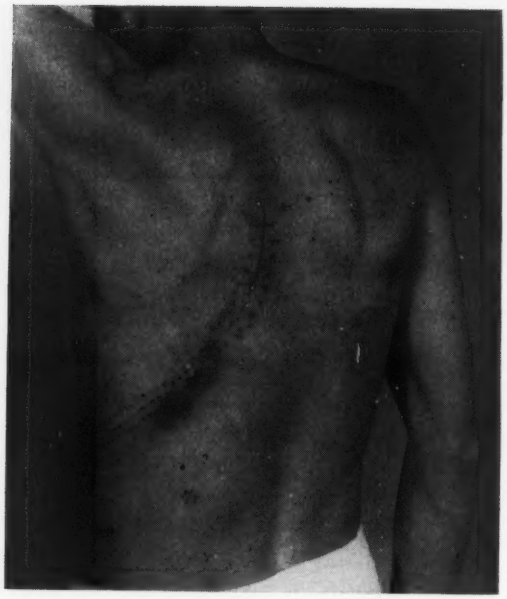

Figure 3. Post-operative view of patient showing the thoracotomy incision following left lower lobectomy and closure of the chest wall defect.

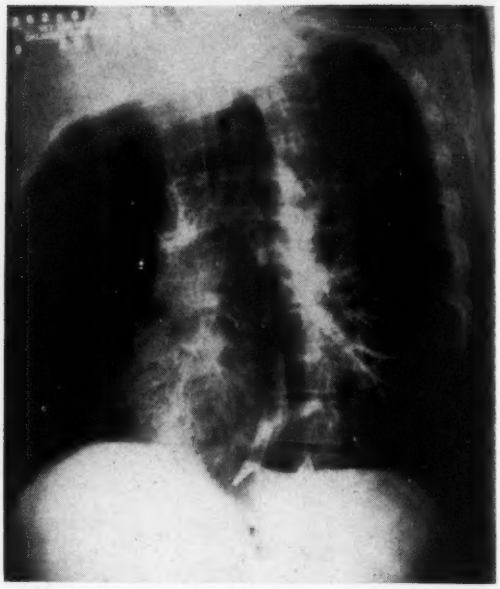

Figure 4. Pre-operative bronchogram (oblique view) showing moderate bronchiectasis of the left lower lobe basilar segments and filling of the peripheral bronchi out to the lung surface at the site of the chest wall defect.

rib was resected. The lining of the defect was excised, and the pleura at this site was freed of adhesions where it was continuous with the superior segment of the left lower lobe. Left lower lobectomy was carried out. The thoracotomy was closed by mobilizing the soft tissue and adjacent ribs above and below the defect. A satisfactory stable closure was obtained (figure 3). Pneumoperitoneum was induced in order to elevate the diaphragm and reduce the size of the pleural cavity temporarily.

The lower lobe specimen showed moderate basilar bronchiectasis. The lung tissue at the site of the hernia was covered by both granulation tissue and bronchial-type epithelium, and two small bronchial communications could be demonstrated. This represented then a chronic lung abscess of the superior segment with broncho-cutaneous fistulization.

The patient recovered without incident, the wound healed primarily (figure 3) and the remaining left upper lobe expanded well to fill the left hemithorax (figure 5).

Discussion

Lung herniae are not common, and the majority of those of clinical significance are acquired and thoracic in location.

Of the others, the cervical herniae are in general congenital and of limited significance. Mediastinal herniation occurs commonly in several situations (such as resection of the opposite lung), but is termed a hernia for purposes of classification only and is ordinarily of secondary clinical importance. Because of the pressure relationships herniation of the lung through a diaphragmatic defect is unlikely, and only one such possible case has been reported.[2] For further description of this group of lesions several general discussions and reviews are available.[3,4,5,6,10]

Thoracic lung hernia represents nothing more or less than a defect in the chest wall of sufficient size and weakness to permit a portion of the lung to protrude when the intrathoracic pressure is increased. Such a defect may occur as a result of infection (tuberculosis, non-specific) or malignancy. Rarely such defects develop spontaneously,[4] usually in the anterior chest near the sternum where the muscular protection of the

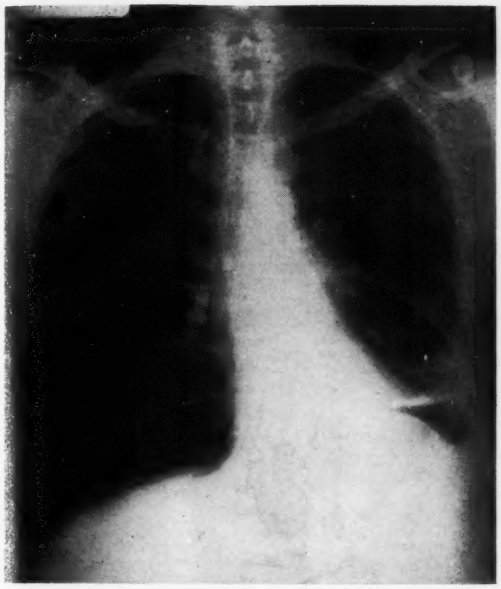

Figure 5. Chest roentgenogram taken two weeks post-operatively showing good expansion of the remaining left upper lobe. Residual pneumoperitoneum induced at the time of surgery can be seen.

thorax is the thinnest. Most commonly, however, thoracic lung herniae are secondary to trauma. Those that develop late after injury or operation have been termed consecutive.

Such diverse accidental injuries as stab wounds,[8] blunt injuries causing rib fractures,[13] gunshot and missile wounds, and goring by a bull[3] have led to defects permitting lung herniation. Intercostal herniation at the site of surgical thoracotomy closure occurs,[1] and it is surprising that it is not seen more often in view of the long incision with potential areas of separation. Surgical resection of rib segments for malignant disease[10] or for purposes of open drainage of the pleural space (authors' case) may, of course, leave such defects.

Operative procedures that require resection of a portion of the chest wall would more frequently result in thoracic hernia were not measures taken to re-establish the thoracic integrity at the time of the primary operation. In this regard the technical considerations in chest wall reconstruction are the same whether the lesion is an established lung hernia or a surgically or accidentally produced defect being treated primarily.

Maurer and Blades,[7] who have presented the largest single series of surgically repaired lung herniae, have emphasized the use of local chest wall tissue in their reconstruction. This includes rib, periosteum and muscle. They stress the importance of closing the defect with bone or periosteum which will produce bone. Such local tissue, with appropriate mobilization, will usually suffice to close the smaller defects (as in the present case) and in those larger ones where the long axis of the defect parallels the ribs.

Where the defect is large or of awkward shape extra-thoracic tissue or material may be required for closure. This may be necessary following the resection of costal cartilages in the extended radical operation for breast carcinoma described by Urban,[11] resection of segments of multiple ribs for chest wall tumors and in the management of close proximity missile wounds of the thorax where wide areas of the chest wall are destroyed.

Fascia lata[11,13] and tantalum mesh[1,9] have been used to provide integrity and stability to the thorax in these situations. A new material which appears most promising for this purpose is Marlex mesh.[12] This plastic mesh is easily sterilized and handled, biologically inert and of satisfactory body and strength. A major advantage over other prosthetic materials is that it apparently does not augment infection or retard wound healing even in the presence of pre-existing though properly drained infection. It is recommended that the coarser and stiffer material be used for thoracic defects and be sutured with stainless steel sutures under moderate tension, thus providing a rather stable chest wall immediately.

Perhaps the most important feature in the management of a lung hernia is the careful evaluation of the underlying lung and pleura. Bronschoscopy, bronchography, sinus tract injection, sputum studies as well as standard roentgenograms of the chest may be required for this. Surgical attack on the chest wall defect should not be undertaken without a concomitant plan of therapy

for the underlying specific or structural disease if present. In the case reported here the appropriate resectional therapy (left lower lobectomy) was carried out for the demonstrated bronchiectasis, chronic lung abscess and broncho-cutaneous fistula. The surgical management of the chest wall defect became almost incidental, despite the fact that the presence of the defect was the patient's presenting complaint.

Summary

A patient with thoracic lung hernia secondary to rib resections and with associated bronchiectasis, chronic lung abscess and broncho-cutaneous fistula is presented.

The relationship of thoracic lung hernia to hernia of the lung in general and to other chest wall defects is briefly discussed.

The use of local thoracic bone, periosteum and muscle for surgical closure of these defects where possible is stressed, and the use of prosthetic material in the larger defects is described.

Careful evaluation and management of any underlying disease of the lung and pleura is essential in the rational approach to the problem.

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Hypothermic Total Body Cardiopulmonary Bypass, Report of 19 Consecutive Cases*

NAZIH ZUHDI, M.D. JOHN MONTROY, M.D.
ALLEN GREER, M.D. JOHN CAREY, M.D.
BILL McCOLLOUGH, M.D.**

WITH THE ADVENT of open heart surgery, divergent schools of procedure have emerged. We have the advocators of normothermic perfusion and the proponents of hypothermic extracorporeal circulation.[1,2,3] Some defend a bubbler and others a filmer. Some recommend the DeBakey and others the Sigmamotor pump. Potassium citrate, acetylcholine, potassium, magnesium and neostigmine, cold, electrical current, and anoxia have been favored to obtain an empty "motionless" heart and to prevent air embolism. Retrograde perfusion of the coronary sinus and direct perfusion of the coronary arteries, with and without cardioplegia, have been tried.

It is apparent that there is ample room for confounding variants. We are not proposing hypothermic perfusion and simple occlusion of the aorta as the final answers to open heart surgery. We are merely presenting a few facts that impressed us favorably during their successful clinical application.[3,4,5,6]

Hypothermic Perfusion

(1) The low oxygen requirements of the body during differential total body hypothermia require comparatively low blood flows that could mimic the cardiac output for that particular temperature. Twenty ccs/min. per kilogram of body weight between intraesophageal temperatures of 20°C and 30°C yield adequate peripheral oxygenation, satisfactory arterial pressures, essentially unchanged electroencephalogram, and urine outputs during hypothermic total body cardiopulmonary bypass. The awakening from anesthesia is immediate and there are no abnormal neurological symptoms or signs.[3,4,6]

(2) The lower flows mean decreased trauma to blood, simpler cannulation and smaller machinery, easier to assemble and clean. The total amount of blood is reduced.

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Pt.	Age Yrs.	Weight Kg.	Diagnosis	Flow Rate ccs/Kg. BW/min.	Total Perfusion Time min.	Duration of Aortic Occlusion min./	Temperature Range During Aortic Occlusion	Final Outcome
1	31	70	Inter-atrial defect	20	38	15*	27°C-30°C	Living and well
2	43	50	Inter-atrial defect	20	41	13*	29°C-30°C	Died 4 mos. post surgery. Autopsy: intact cardiac repair and acute necrotizing pancreatitis.
3	4	16.3	Tetralogy of Fallot	20	69	22**	26°C-30°C	Died on 2nd P.O. day after doing well. Autopsy: Partial disruption of I.V. repair.
4	14	35	Tetralogy of Fallot	20	121	66**(20, 23, 7 & 16)	27°C-30°C	Living and well
5	4	14.5	Pulmonary valvular and infundibular stenosis	20	124	0**	23.5°C-31°C***	Living and well
6	11	43	Inter-atrial defect. Expl. for pos. pulm. stenosis.	20	75	24**(18, 6)	27°C-30°C	Living and well
7	35	43	Mitral commissurotomy	20	38	18**	26°C-30°C	Living and well
8	8	19.3	Tetralogy of Fallot.	20	154	90**(15, 15, 15, 15, 15, 15)	25°C-26°C	Died on 3rd P.O. day. Autopsy: intact cardiac repair. Severe bil. pneumonia
9	9	25.56	Inter-atrial defect. Pulmonary stenosis.	20	58	12**(6, 6)	28°C-29°C	Living and well
10	5	14	Inter-vent. septal defect. (pulm. hypertensive)	20	121	40**(16, 8, 16)	24°C-25°C	Living and well
11	31	59.05	Inter-atrial defect	20	88	18**	24°C-25°C	Living and well
12	14	27.64	Inter-vent. defect. (pulm. hypertensive)	20	85	24**(18, 5)	23°C-26.5°C	Living and well
13	42	51.77	Pulm. stenosis and Infundibular stenosis	20	100	25**(5, 15, 5)	28°C-31°C	Living and well
14	7	18.96	Tetralogy of Fallot	20	116	27**(15, 7, 6)	23.5°C-24.5°C	Living and well
15	6	16.5	Tetralogy of Fallot	20	106	18**	22.25-24°C	Living and well
16	12	59.12	Pulmonary Stenosis	20	46	8*	28.5°C-29°C	Living and well
17	9	16.4	Inter-vent. septal def. pulm. hypertension.	20	80	28(3, 19, 16)*	24°C-26°C	Living and well
18	52	38.7	Mitral Stenosis and insufficiency	20	69	11*	29°C-31.2°C	Living and unchanged from pre-op. condition. Condition of the valve was such that no repair was performed
19	25	58.58	Inter-vent. septal def.	20		34** (17, 17)	25.1°C-25.2°C	Living and well

/Figures between parentheses indicate single periods of aortic occlusion.
*Ventricles continued to have regular rhythm.
**Ventricular fibrillation that responded to one electroshock of 110 volts.

TABLE I. Data on hypothermic total body cardiopulmonary bypass on 19 consecutive cases.

(3) Lengthy perfusions are permitted and thorough evaluation of complicated defects accomplished.[6] Sutures are deliberately and accurately placed and patches precisely fashioned. The aim should be a lifetime cure. The continuation of perfusion for sometime after closure of the cardiac chamber may be partially responsible for the excellent behavior of the heart.

(4) Simple prolonged aortic occlusion is the easiest answer to the obtainment of an empty "motionless" heart and to prevent air embolism.[6] The electrocardiogram has shown no patterns of ischemia and the gross and microscopic detailed examination of three hearts revealed no changes compatible with necrosis, liquefaction or fibrosis.

(5) Table I summarizes 19 consecutive patients who had hypothermic cardiopulmonary bypass for open heart surgery and stresses some of the advantageous features of this technique.

Comment

Heat exchangers have been experimentally and clinically interposed in heart-lung machines for some time.[1,2,3] The simplicity, availability and efficiency are the main tributes of the one we use and has been described in detail elsewhere.[5]

The disadvantages of deadly ventricular fibrillation, limited time of venous inflow stasis, tendency to bleeding and rewarming shock of external hypothermia, and the drawbacks of high flows and increased bronchial and coronary sinus returns of normothermic perfusion are eliminated by hypothermic extracorporeal circulation. In addition, prolonged simple clamping of the aorta becomes feasible and provides the simplest way to obtain an empty "motionless" heart and to prevent air embolism. The findings of Gott et al[7] that high energy phosphates, glycogen and lactic acid of the myocardium are minimally changed during hypothermia arrested hearts corroborate our results.

ADDENDUM

Since the submission of the manuscript, this technique of open heart surgery was performed on 18 additional service and private patients without a mortality. Up to the date of proof reading (February 3, 1960), there have been 29 consecutive survivals. The completeness of the repair and back to normal life behavior are most noteworthy.

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The Present Status of the Internship

The internship in this country had its beginning in institutional apprenticeships, originating about 160 years ago. Official recognition first came in 1904, when the American Medical Association established standards for the internship as a part of the training of a physician. Facilities for intern training first exceeded the number of medical graduates in 1926, and stayed slightly greater than the number of graduates until just after World War II, when available positions began to increase rapidly. The number rose from about 8,000 in 1945 to 12,649 last July, at which time there were 6,860 graduating seniors from United States medical schools.

In spite of the increase in internships, the percentage of positions filled has remained at 83 per cent. Obviously only about 55 per cent of these were filled by graduates of United States medical schools. About 25 per cent were taken by graduates of foreign schools. The number of foreign graduates serving internships first became significant in 1950 and has increased rapidly since then. This rise was partly due to the passage of the United States Information and Educational Exchange Act of 1948 which facilitated the securing of visas for educational purposes. Now there is a good possibility that the number of foreign graduates available for internships will decrease because all hospitals listed for internship have agreed to accept no foreign student after July 1, 1960 who has not been certified by the Educational Council for Foreign Medical Graduates.

All of these facts show one great problem connected with the internship: a shortage of interns. Now an institution which is rooted deeply by tradition, and fixed by law, is the target for all types of recommendations for change, including abolishment.

The internship has several purposes. First it should bridge the gap between didactic instruction and practical application of the principles of medicine. Secondly, the intern learns to accomplish certain necessary procedures, to exercise judgment and make decisions, all in a setting and under circumstances where he does not have to bear the full and sole legal responsibility for his acts. Thirdly, the intern year gives the serious student of medicine a chance to decide what his future field of endeavor will be.

It must be realized that at least five parties determine the character of an internship. They are the medical student, the hospital, the Council on Medical Education and Hospitals, state boards of licensure and the medical schools. The first two parties are most directly involved and it is the balance between these two which is most important. The intern owes the hospital his loyalty and a desire to work and the hospital owes the intern educational opportunity and protection from spending too much time in tasks that have ceased to be of value in his career. This balance often is upset, with both parties being the offender.

Many solutions have been suggested for the internship problem, none of which are agreeable to everyone concerned. The least practical is abolishment of the internship, which is untenable just for the licensure problems involved.

Mark R Everett

The Normal Radiology of Old Age and Its Borderlines

ERNEST LACHMAN, M.D.

Since graduating from the Breslau Medical School in Germany in 1924, Ernest Lachman, M.D., has been certified by the American Board of Radiology. He is Professor and Chairman of the Department of Anatomy and Consultant Professor of Radiology at the Universiy of Oklahoma School of Medicine.

Doctor Lachman is a member of the American Association of Anatomists, the American College of Radiology, the American Association of Physical Anthropologists, the Society of Experimental Medicine and Biology and the Gerontological Society.

RADIOLOGY has added a new dimension to the time honored field of human anatomy by contributing a vast amount of information on normal age changes from infancy to senescence in tissues and organs that are accessible to the roentgenologic approach. For the older age groups a frame of reference can be established against which one can evaluate pathologic alterations that are the concomitant of old age. Here, as so often in radiology, the "borderlands of the normal and early pathological" (Koehler) have not been clearly defined. But most radiologists have accumulated, almost intuitively, a storehouse of visual impressions as to variations of old age, such as the increased translucency of lungs and bones, the greater number of vascular calcifications, alterations in the configuration of the spinal column and various joint changes.

While hardly any systematic presentation of these changes in the roentgenogram has been undertaken, there is nevertheless a considerable amount of information scattered through the roentgenologic and gerontologic literature. A brief review of this material seems indicated.[1]

Thoracic Cage

There is great need for vertical studies which follow healthy individuals through their life span by means of roentgenograms of the chest in one or two projections. With modern technique radiation dosage can be restricted to a minimum and the gonads protected, so that the obstacle of radiation hazard can be overcome. Until such longitudinal studies have been done, we have to be satisfied with comparative statistical appraisal of average values for different age groups. Such cross-sectional studies have been undertaken by various authors. They show that with advancing age there is a significant increase in the distance between the thoracic inlet and the dome of the diaphragm, i.e., in the roentgenographically observable height of the lungs. The maximal increase is present in the seventh decade. Concomitant with this increase and possibly causing it, flattening and lowering of the diaphragm is observed. Some investigators also demonstrate a statistically significant increase in the antero-posterior diameter of the chest or thoracic depth from the fourth decade on. Thoracic width remains stationary through-

out life. Also noted is an increase in the downward slope of the ribs, particularly in the oldest age groups. Well known is the progressive calcification of the costal cartilages with the interesting sex difference that calcification of the first costal cartilage advances much more rapidly in the male than in the female where the terminal stage of almost complete calcification is only rarely attained.

Lung and Trachea

It can be assumed that the enlargement of the thoracic cage in height and depth leads to a distention of the lung with some degree of emphysema. Indeed numerous authors have stated that emphysema is almost universal in old people. It is questionable to what extent the roentgenogram depicts minor degrees of senile emphysema. There is lack of correlation between the roentgenogram on one hand and the clinical and postmortem findings on the other. Many variables govern radiographic translucency of the lungs. These include technical factors, the depth diameter of the thorax and the degree of development of extra- and intrathoracic soft tissues. Thus increase in the over-all translucency of the lungs due to atrophy of the interalveolar septa might be counteracted by lengthening of the depth diameter of the chest, by increased filling of the blood and lymph vessels due to pulmonary congestion, and by fibrosis or bronchiectasis accompanying emphysema. The great number of variables precludes quantitative evaluation of the degree of emphysema from the roentgenogram. Other roentgenograhpic signs of emphysema, frequently found in old age, are depression and flattening of the diaphragm and restriction of respiratory excursion. There seems to be a positive correlation between advancing age and the percentage of roentgenologically detectable emphysemas so that one might almost be inclined to regard the early signs of emphysema as a physiological concomitant of senescence. Here, as in other field of geriatric roentgenology, it is difficult to draw the line between the normal involutionary process and degenerative pathology. Attention should be called to the intensification of pulmonary markings in people over 60 years of age. This intensification may be vascular or bronchial in character and may be due to pulmonary hypertension and beginning congestion or interstitial fibrosis and chronic bronchitis. A combination of all these factors is of course possible. Chronic inflammatory and cicatricial processes may lead to enlargement of the hilar shadows in advanced age. These alterations seem to be due to anthracotic and silicotic hilar sclerosis. This may occur in elderly inhabitants of industrial centers, even without occupational exposure to coal dust, since the longer life span allows for the accumulation of greater amounts of foreign material and consequent reaction to it. Frequently noted has been the gradual descent of the tracheal bifurcation with advancing age, which in old age may be as low as the seventh thoracic vertebra.

Cardiovascular System

Thoracic Aorta. There is a progressive elongation and dilatation of the aorta in the older age groups, a change which is due to loss of elasticity in the vessel wall. Consequently there is an increase in the size of the aorta as determined by roentgenographic measurements. It is also known that with advancing age more and more of the ascending aorta becomes marginal along the upper right arch of the cardiovascular silhouette in the posteroanterior view, resulting often in increased convexity of this part of the vascular contour. The aortic knob protrudes farther cranially and to the left. The left lateral contour of the descending aorta is visible to a greater length and may intersect the cardiac shadow in the region of the left auricle. Oblique views demonstrate the greater convexity of the whole thoracic aorta. Its shadow may be denser and the aortic knob frequently shows calcification.

Heart. The described changes in the aorta effect the roentgenographic appearance of the senile heart to a considerable degree, since it has to adapt itself to the available space in the mediastinum, in part preempted by the tortuous and enlarged aorta. As a consequence the heart frequently becomes more transverse. The final size, shape, and position of the "normal" senile heart is the resultant of a number of partly conflicting factors, such as the degree of

aortic dilatation, level of diaphragm, thoracic shape, presence of senile emphysema, obesity, and hypertension. Most statistical studies agree that with increasing age there is a slight increase in actual heart size, especially due to enlargement of the left ventricle. If hypertension and arteriosclerosis are marked, then the heart assumes aortic configuration which is characterized by prominence of the aortic shadow on both sides, prominence of the left ventricular contour and a deepening of the cardiac waist.

The senile atrophy of the heart with marked reduction in cardiac musculature, that is so often diagnosed by pathologists as a characteristic sign of advanced age, is generally not reflected in the roentgenogram during life because of the dynamic and variable conditions of filling. Aortic calcifications, which are a common accompaniment of old age, have been mentioned. Rarer are demonstrable calcifications in the coronary arteries and the mitral and aortic valves. The clinical value of such findings is debatable. There seems to be no correlation between the roentgenologic finding of aortic or coronary calcification and the functional effectiveness of the circulation.

Other Blood Vessels. Other vascular calcifications are also more common in the elderly. Well known is the roentgenogram of the calcified abdominal aorta, particularly on lateral views. This condition is the best clinical sign of the atherosclerotic type of arteriosclerosis and should be more frequently recognized in clinical surveys for arteriosclerosis. Rarer are calcifications in branches and sub-branches of the abdominal aorta, such as in the splenic artery. They occasionally have been mistaken for foreign bodies. More frequent are calcifications in the arteries of the extremities which appear as two main types, that of the intima and media. The intimal type is irregular with scattered discrete plaques and is often confined to certain parts of an artery and to certain sections of the vascular circumference. It is most easily demonstrated by radiographic soft tissue technique in the lower third of the femoral artery. These calcifications are a sign of arteriosclerosis and as such occur more frequently in conjunction with occlusive arterial thrombosis than with patent arteries; the roentgenographic demonstration of this type, therefore, has clinical significance.

By contrast, in calcification of the media, also known as Moenckeberg's sclerosis, the calcifications are regular, diffuse, and of fine grain and affect in general the whole circumference of the artery. There is a tendency for the calcium to accumulate in rings and to be wide-spread in character. This is the "pipe stem" type of calcification which may occur in old age without leading to obliteration of the involved vessel. It is often only an incidental finding accompanying senescence. In younger people it is an indication of disturbances of calcium metabolism.

Calcification of the internal carotid artery deserves special mention. The calcified artery, particularly if tortuous, is projected into the hypophyseal area on lateral views, which may lead to the erroneous diagnosis of calcified pituitary tumor.

The importance of calcifications of thrombi in the veins of the vesical, uterine and prostatic plexuses lies mainly in their being mistaken for ureteral or vesical calculi. The occurrence of these phleboliths is normal in older age groups.

Gastrointestinal Tract

The digestive system of the aged seems to display less wear and tear than most other systems of the human body and consequently seems capable, if not locally altered by malignancies or toxic and infectious processes, of functioning beyond the ordinary life span. Thus geriatric roentgenology of the gastrointestinal tract furnishes only a few relevant data that affect our standards for the normal adult person.

Esophagus. As far as the esophagus is concerned, the increased frequency of hiatus hernia in older age groups has often been noted, particularly in the female sex. It is particularly the sliding type of hernia without shortening of the esophagus that is apt to be found in the elderly. Loss of elasticity in the tissues around the hiatus with advancing years is apparently the cause of the condition. While there is a whole series of symp-

toms referable to hiatus hernia, it should be realized that it may be asymptomatic and only an incidental finding in a carefully performed roentgenologic examination of the gastrointestinal tract.

Stomach. So far no changes in the roentgenographic anatomy of the stomach accompanying old age have been reported in the literature. Size, shape, position and tonus of the organ seem to remain unaltered. Although there is considerable evidence that in the elderly histologic changes occur in the mucosa of the stomach, pointing in the direction of an atrophic gastritis, and that the incidence of achlorhydria increases, none of these conditions is apparently accessible to roentgenographic demonstration. The rate of gastric evacuation after a barium meal does not seem to be affected by old age.

Small Intestine. It is known that the cranial most point of the duodenum moves caudally in both sexes at a rate of half a vertebra per decade. But since the duodeno-jejunal flexure is relatively fixed, the vertical spread of the organ decreases with age. The following abnormalities have been found in a roentgen study of the small intestine in people over seventy years of age without clinical symptoms: segmentation of filling, hypertonus, hypotonus, and spasticity of portions of the intestine, hypermotility or delay in emptying. These alterations have been explained on the basis of arteriosclerotic interference with the blood supply of the intrinsic nerve plexuses of the intestine.

Colon. Roentgenologic evidence does not support the frequently expressed claim that the colonic musculature becomes atonic with advancing age and that the time of passage is delayed. But only insufficient data on the normal colon in the aged are available. Diverticulosis of the colon is a common and often incidental finding in the roentgenologic examination of older people, belonging to the borderline field of the normal.

Biliary and Urogenital Tracts

There are neither horizontal nor vertical studies available which evaluate age changes in the size, shape, and position of the gallbladder and kidney on a statistical basis. One author found considerable distension of the fundus of the senile gallbladder. On the other hand excretory studies on the gallbladder in old age seem to prove that if the biliary tract escapes pathologic alterations, its motility does not become impaired. The incidence of gallstones progressively increases with old age. They were found by one author in 70 per cent of all cases over seventy years of age not all of which of course would be demonstrable on the x-ray film. The "normal" degenerative changes, that take place in the human kidney in the process of aging, do not seem to be reflected in either a decrease in the size of the kidney shadow or in a slowing of the excretion of the contrast material in intravenous pyelography. Prostatic enlargement and diverticula of the bladder can be demonstrated radiographically by the proper cystographic procedures.

Skeleton

The skeleton of the aged displays a number of roentgenologically discernible signs of involution. Osteoporosis in older people seems to be an almost physiologic condition, caused mainly by impaired activity of the osteoblasts with resulting inadequate production of bone matrix. The disturbance manifests itself as postmenopausal osteoporosis in women past fifty and as senile osteoporosis after the age of sixty-five, the two conditions being apparently similar in origin and pathology. Anatomically, osteoporosis of old age is characterized by decrease in the thickness and number of bony trabeculae with widening of the intertrabecular spaces. The compact matter disappears more slowly. In milder cases osteoporosis is limited to spinal column and ribs. Further advance results in involvement of pelvis and proximal femura, leading finally to inclusion of the whole skeleton. It requires the disappearance of fairly large amounts of calcium, before the diagnosis can be made roentgenographically. The minimum amount of decalcification necessary for roentgenological visibility has been variously given as 20-60%. It has been estimated that more than half of all women over the age of fifty and more than half of both sexes past sixty show a sufficient degree of osteoporosis to be roentgenologically demonstrable.

Early roentgenographic signs of senile osteoporosis may not be easily recognized; in fact minor decrease in bone density may be ascribed to technical variations such as over-exposure or over-penetration. In more marked cases the trabeculae are clearly scarcer and thinner than normal, with widening of the trabecular meshwork. Later the cortex also becomes thin and lamellar with increase in the width of the medullary cavity, and it apears as if drawn with a fine pencil. Finally, trabecular structure may become almost invisible and the bone takes on a glass-like appearance.

Senile osteoporosis is most easily demonstrated roentgenographically in the spinal column. The bone texture appears as previously described, with the added feature of asymptomatic collapse of one or more vertebrae. The "codfish" appearance of the spinal column with over-all decrease in density, with ballooning of intervertebral disks, and biconcave deformity of the vertebral bodies offers a picture that is often in startling contrast to the scarcity of symptoms. Clinically undiagnosed fractures occur in as many as 25% of all cases. The lower thoracic and upper lumbar segments of the column are most commonly affected by these changes, while wedging of the vertebrae as a result of osteoporosis involves mainly the mid-thoracic vertebrae and may lead to marked kyphosis of this region. This osteoporotic type of senile kyphosis has to be differentiated from ordinary kyphosis of old age. While the site of both deformities is the same—the mid-thoracic region—their anatomy is quite different. In the osteoporotic form the kyphosis is the result of a wedge-shaped collapse of one or more vertebrae, while in the ordinary type of deformity is brought about by degeneration and partial disintegration of the disk with preservation of the quadrangular form of the vertebral body. For static reasons mainly the anterior portions of the intervertebral cartilage are involved, which in the final stage are completely replaced by a bony bridge between adjacent vertebrae. In the oldest age groups there is often a combination of the two types.

In the pelvis and upper femur the disappearance of part of the spongy matter may result in coarsening of the functionally most important trabeculae which—due to thinning of the cortex—stand out quite distinctly. Structural weakening of the bone often leads to decrease in the angle between the axes of the neck and shaft of the femur with advancing age. This, and the changes in the spinal column, are the reasons for decrease in height in older individuals.

The other long bones as well as the skeleton of hand and feet may display similar changes consisting of cortical thinning, widening of the trabecular meshwork and over-all decrease in bone density. The roentgen signs of senile osteoporosis may remain stationary for many years and may be unaccompanied by any clinical symptoms, as stated before.

A second group of bony changes, associated with advancing age and on the borderline of the normal, are alterations along the articular surfaces of long bones, vertebral column and ribs. They are the result of degenerative changes in the cartilage which, on the basis of chronic wear and tear and decreased blood supply, loses its elasticity. The bony changes comprise reactive ligamentous ossifications and new bone formation at the capsular attachment of the joint. Anatomically and roentgenographically the process assumes the form of marginal osteophytes, spurs and lipping; clinically it is described as senescent, degenerative, hypertrophic or osteoarthritis. The condition seems to be universal after the age of forty-five and progresses with advancing years. The heavier constitutional type is apt to acquire these changes earlier than the slender type.

There are hardly any roentgenologic studies available on the distribution and frequency of this condition throughout the skeleton. But we know that the articular surfaces of the vertebral column, of pelvis and knee joint are most apt to reveal roentgenographic changes, although the joints of the upper extremity, likewise, do not escape. In the knee joint, in addition to narrowing of the joint space, spur formation and lipping, we find occasional asymptomatic calcification of the menisci. Spurring on the calcaneus also is stated to be more common

in older age groups. Quite interesting is the observation that the frequency of sesamoid and accessory bones increases with advancing age.

Head and Neck

Numerous are the changes of old age in the roentgenogram of the skull. Senile atrophy is quite common, leading frequently to a "washed-out" appearance of the calvarium, which may be uniform or spotty and which has to be differentiated from ostitis fibrosa cystica and the early stages of Paget's disease. In one series two-thirds of the aged subjects showed roentgenographic evidence of minimal to moderate osteoporosis of the skull. The sella turcica is one of the first regions to reveal these changes. The dorsum sellae appears thin and translucent with narrowing also from side to side, alterations that have to be distinguished from localized pressure erosions. The over-all glass-like translucency of the sellar area contrasts sharply with the frequently present calcifications of the superimposed internal carotid artery. As far as the paranasal sinuses are concerned, some diminish in size in old age, particularly the maxillary sinus, while the frontal and sphenoidal sinuses increase in extent.

In regard to the roentgen pattern of the diploic veins, the literature contains conflicting statements. While one group of authors speaks of regression with age, others mention enlargement and widening of the diploic canals. The depressions caused by arachnoid granulations become progressively larger with advancing age although there are marked variations from individual to individual.

In contrast to the common appearance of osteoporosis of the senile skull, a hyperostosis of the cranial bones is rather rare. This is located in the anterior cranial fossa, involving the frontal bone, and may be diffuse or circumscribed and nodular. It represents a condition on the borderline of the pathologic and occurs mostly in women past middle age.

The cranial sutures are as variable as other skull markings mentioned so far, although there is a typical picture represented by complete closure of all sutures in old age. In general the coronal suture is almost invariably closed in older age groups, the sagittal suture in women past fifty and in men past sixty; the lambdoid suture may remain open throughout life in a fair sized number of cases and this may occasionally happen also in other sutures. Where sutures are obliterated, their sites may be indicated by a line of denser sclerotic bone.

Omitting dental and alveolar changes from this discussion, one normal finding in the skull of the aged remains to be mentioned, i.e., the calcifications in the pineal body, in the falx cerebri, in the margins of the diaphragm of the sella turcica, in the free edges of the tentorium cerebelli and posterior to the dorsum of the sella and in the choroid plexus, in addition to the calcifications in the internal carotid artery mentioned previously. While calcifications in the pineal body may occur in younger years and even in adolescence, they reach a frequency of 78% in people past sixty. They have been found in 55% of all individuals past twenty-five.

Age changes in the form of laryngeal calcifications were known even before the roentgen era. While ossification of laryngeal cartilages starts around the age of twenty, it progressively increases, reaching its maximum extent around the sixty-fifth year. Calcification may also involve the tracheal rings. In the larynx, as so often, there are many variations in the aging process even to the extent of complete absence of calcifications in the older individual. One author found that there is a definite relation between the degree of ossification of laryngeal cartilages and the presence of mineral matter in the wall of the aorta.

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FOOTNOTE

1. For a detailed bibliography see the author's paper listed under "References."

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Etiology of Peptic Ulcer Disease

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Doctor Jerry Bressie: The patient for this morning is Mr. M. P., a 20 year old white male who was admitted on 11-10-59, complaining of vomiting coffee ground material and having tarry stools on the day of admission.

PRESENT ILLNESS: This 20 year old white male filling station employee first became ill at the age of 12 years, having sharp, severe periumbilical pain which disappeared after about one week, but which reappeared, and has continued to be intermittantly present since the age of 16 years. These were not typical "ulcer pains" but nonetheless the patient had a perforation of an anterior duodenal ulcer at the age of 18 years, which was repaired at University Hospital. He was then placed on management consisting of frequent feedings and antacids which he stated that he followed religiously. Nevertheless in April, 1959, he had the acute onset of hematemesis which required the replacement of four units of blood. He was treated conservatively and remained on an ulcer program but continued to have the periumbilical cramping every two or three weeks. He did, however, note transient relief of this with the intake of alkali and antispasmodics. Approximately 4:00 p.m. on the day of admission he had the onset of nausea and promptly vomited approximately one pint of coffee ground material, shortly followed by the passage of black feces. On his arrival at University Hospital he vomited blood a second time. The past history and review of systems is non-contributory.

SOCIAL HISTORY: The social history is of interest in that the patient quit school in the 10th grade. He has been employed at many jobs, usually as a farm laborer and more recently as a filling station attendant. It is noteworthy that he has had a bleeding episode or exacerbation of the ulcer pain following each new employment. He feels that he is a failure in life and now aspires to employment of a more technical nature, which would require a period of special training which he cannot undertake due to the responsibilities of a home and family. He feels that he is now destined to remain in a more or less menial job for the remainder of his life.

PHYSICAL EXAMINATION: The blood pressure on admission was 110/70; pulse 120 per minute; respiration 16 per minute.

He was quite pale and appeared acutely ill. The significant physical findings were otherwise limited to the abdomen, where no guarding or rebound tenderness was elicited. Bowel sounds were present. The liver was down one cm. beneath the right costal margin but no other viscera or masses were palpable.

LABORATORY STUDIES: Laboratory studies on admission revealed a hemoglobin of 9.5 gms.% with a hematocrit of 32%. The white blood count was 15,700 per cubic millimeter with a differential of 47% polymorphonuclear leukocytes, 43% lymphocytes, 4% monocytes, 3% eosinophils and 2% basophils. Bleeding and clotting studies were normal as was the urinalysis. A stool examination on admission was positive for blood, however by the sixth hospital day the stools had become negative. The alkaline phosphatase was 1.7 Bessie Lowery unit, transaminase studies were within normal limits, BUN was initially 33 mgs.% but by the sixth hospital day, had fallen to 14 mgs.%. The fasting blood sugar was 100 mgs%. Calcium and phosphorus determinations were within normal limits. The gastric analysis showed a total volume of 175 cc. with a free acid of 70 degrees and a total acid of 80 degrees.

HOSPITAL COURSE: The patient was treated initially with whole blood transfusions and following this showed no further evidence of significant bleeding. He has subsequently been given frequent feedings and interval antiacids. He is at present asymptomatic and is eager to leave the hospital.

Doctor Wyman Ewing: A single PA view of the chest shows a normal heart and lungs and the visualized osseous structures appear normal. We also have an upper GI series which surprisingly enough shows a normal esophagus, stomach, duodenum and proximal small bowel. These films are perfectly normal.

Jack D. Welsh, M.D.: Any attempts to present an orderly review of the theories regarding the etiology of peptic ulceration are destined to frustration. This is in part due to the great quantity of recorded experimental data and philosophical ideas and to the conflicting interpretations of these data. In addition, difficulty is encountered in the clear separation of "primary causes" from "contributing factors." With these limitations in mind, I shall attempt to outline the major theories of causation that have, at one time or another, been popular in the medical world.

The conditions considered a possible cause of, or contributing factors in the genesis of peptic ulcers may be separated into 12 general categories. I would like to review these briefly before we consider present-day theories. No attempt will be made to present all of the available data concerning each of these, or to mention all of the individuals concerned.

Review of Theories

1. *Vascular:* Rokitansky, as early as 1849, thought the etiology of peptic ulceration of the upper intestinal tract was due to hemorrhagic erosion. The idea of vascular embolism, thrombosis, and infarction causing the ulceration was proposed by Virchow in 1853. Peculiarities of the blood supply, atherosclerotic changes, and more recently, arterial spasm, and alteration in shunt systems have also been implicated.

2. *Mechanical:* Aschoff suggested that mechanical factors were of importance in producing ulcers. He proposed that such ulcers occurred along the lesser curvature in the so-called Magenstrasse, in areas which are most exposed to traumatization from the acid and gastric contents. Other mechanical factors that have been proposed are: chronic compression of the abdomen, blows to the abdomen, rough foods, spasm and hyperperistalsis.

3. *Infection:* Various observers have considered foci of infection to be important. This thesis was particularly championed by Rosenow from 1913 to 1923. Recently, less credence has been given to the focal infection theory; however, the frequent association of ulcers and respiratory infections is well recognized.

4. *Chemical:* Cohnheim, as early as 1880, and Regal in 1886, proposed the idea of hydrochloric acid causing ulcers. In the early 1900's, many workers such as Hurst,

Stewart, and Sippy, stressed the role of hydrochloric acid and pepsin. Since these factors are still considered to be important, they will be discussed later in more detail.

5. *Neurogenic:* The relationship of intracranial lesions and acute ulceration of the upper intestinal tract has been noted by such men as Rokitansky, Voderahe, Cushing, and others. These cases, however, form a special group and probably have little or no relationship to the type of ulcer being discussed today. Extensive data have accumulated on the influence exerted on gastric secretion and motor function by the accelerator influence of the vagus and the inhibiting influence of the sympathetic nervous system. Von Bergmann felt that a functional disharmony might exist between the vagus and sympathetic nerves thereby causing spasm of the musculature and vessels of the stomach and duodenum, this in turn leading to submucosal ischemia, erosion, and finally to ulceration.

6. *Allergy:* The ability to produce allergic lesions in the intestinal tract of animals and the reduction of symptoms in some patients by elimination diets, have caused some to believe that peptic ulcers have an allergic origin. Although this may be a contributing factor in rare cases, its importance in the genesis of the majority of ulcers is doubtful.

7. *Constitutional and predisposing factors:* Much has been written concerning the "type" of individual who develops an ulcer. This will be discussed later.

8. *Gastritis:* The role which gastritis plays in the pathogenesis of ulcer remains highly controversial. Erosion and acute ulcers of the stomach are common accompaniments of chronic gastritis. Whether this pertains in duodenal ulcer has not been established. Conversely, however, there is ample proof that intermittent pylorospasm and pyloric obstruction may bring about secondary gastric changes.

9. *Nutritional deficiency:* Diets high or low in protein, high or low in carbohydrate, or diets deficient in fat or vitamins have been blamed for the production of peptic ulcers. The only definite conclusion is that animals and man can develop ulcers on abnormal diets.

10. *Endocrine:* The possibility of endocrine imbalance leading to ulceration of the gastrointestinal tract is not a new consideration. It has received new impetus over the last few years with the demonstration of ulcerations associated with tumors of the pancreas and other endocrine glands. Recent studies, beginning with Selye's introduction of the "general adaptation syndrome," would suggest that the adrenal glands may participate in the pathogenesis of peptic ulcer.

11. *Intragastric mucosal irritants:* Experiments with corrosive substances in animals, the high incidence of peptic ulceration in Abyassinia and India where the natives' diet is generously seasoned with pepper, and many other such observations have led some to the conclusion that irritants in the food are an important factor in the genesis of ulcers. It is questionable, however, whether the experiments in animals are comparable with the conditions in humans. Also, many of the groups whose diets contain large amounts of condiments have associated nutritional deficiencies.

12. *Occupational:* I have mentioned this only for completeness since Doctor Honska will discuss the influence of occupation in more detail.

Modern Concepts

Even with our present "sophisticated" approach to peptic ulcer disease, the literature is filled largely with information obtained through a gastric tube or from an x-ray machine. Since the x-ray evidence of ulceration of the duodenum of the patient presented today was debatable, I think it is relevant to quote from an article, "The Trouble We're in Over Duodenal Ulcer," by Eddie Palmer.[1] "Upsetting as it may be, it is necessary to admit that the reason is that, of the complex total-patient disease which 'ulcer' is, only the little crater can be measured by an objective device, a device which doesn't require much thinking—or at least much assumption of critical diagnostic responsibility—on anybody's part. Professional insecurity being what it is, it sometimes seems best to wait for the x-ray machine to tell what the prob-

lem is or even whether the patient is sick or not. This misses the point of ulcerative disease, because it really doesn't matter very much whether the little crater is there or not. Among the things that the patient with ulcerative disease is likely to show are narrow subcostal angle, a high arched palate, and a crater in the duodenum—but not always. And, because anatomy permits it, the crater comes and goes—not as the indication of a disease, but in response to the disease. The patient isn't sick because he has an ulcer, but has an ulcer because he is sick."

Most workers in the field today question whether just one factor is responsible for all peptic ulceration. Rather, it is felt that an interplay of several factors, varying in different persons and even in the same individual under changing circumstances contributes to the production of peptic ulcer disease. These factors can be divided into two main groups, each with their own subdivisions. (1) Local factors. (a) Eroding factors. (b) Local tissue resistance, (defense mechanisms, and ability to heal). (2) General factors. (a) Physiologic. (b) Genetic or constitutional proclivity. (c) Psychologic. (d) Ecologic or environmental. Present evidence would suggest that these may be interrelated, at least to some extent, by neural, and hormonal factors.

(1) *Local*

a. *Eroding factors:* It is well known that if there is no acid there is no ulcer.[2,3] Some feel that the acid is probably more important than the pepsin in the production of the lesion,[3] however, both are necessary. Considering duodenal ulcer patients as a group, they are hypersecretors, have increased gastric acid and pepsin, respond more than non-ulcer patients to secretory stimulating agents, and their night secretion is greater than in normals or patients with gastric ulcers.[3-7] Taken individually, however, some duodenal ulcer patients secrete less than normal persons,[3] even when the ulcer is in an active phase.[8] In the duodenal ulcer patient who has hypersecretion, it is continuous and the stomach doesn't get a "breathing spell" or rest from this high acid-pepsin bath. Experimentally it has been shown that the continued dripping of excess acid into the stomach of dogs depresses the neutralizing ability of their stomachs.[9] We do not know whether this happens in man; however, it is possible that in some patients the continued hypersecretion of acid may do the same. A look at the other side of the coin reveals that the magnitude of the hypersecretion is not enough to be the sole cause of the ulcer.[8,10] The secretory activity of patients with duodenal ulcer is the same whether or not there is an active ulcer. In other words, the ulcer can come and go while the stomach is at the same high secretory level.[3,8,11,12] Therefore, if the secretory level were the sole factor in producing the ulcer, it should wax and wane as does the x-ray evidence of the ulcer crater.

b. *Local tissue resistance, defense mechanisms, and ability to heal:* Gastric biopsy of another area of the stomach at a time when an active ulcer is present will heal in about two days without producing an ulcer. What prevents the healing of one break in the mucosa and not the other is an interesting question on which to speculate. This has been thought to be anatomical and related to the trauma caused by the passage of food or to the jet of gastric juice passing from the stomach and striking a localized area in the duodenum. There has also been shown to be a gradient of susceptibility to the eroding effects of an acid-pepsin mixture in the digestive tract.[10] Vascular changes may influence this local tissue resistance and, as we mentioned, earlier workers felt that this was related to endarteritis and other changes leading to ischemia. However, Wolf and others have shown that hyperemia precedes the production of petechiae, increased fragility, and ulceration.[13] Recent work with microradiographs has increased our knowledge of the shunting systems in the mucosa and submucosa in the intestine. It has been shown that vagal stimulation closes these shunts and can increase blood flow to the mucosa thereby producing plethora and possibly decreased tissue resistance. If this is a factor, why it occurs only in such a localized area has not been explained, but apparently vascular changes may be important.

(2) *General*

Since any division of these factors is ar-

tificial because of their inter-relation, it will be necessary to refer back to the local conditions in the discussion of general factors.

a. *Physiologic:* The upper intestinal tract appears to have a proclivity to respond to different noxious stimuli by ulcerating.[14] This has been well documented both experimentally and clinically. First, acute ulceration of the upper intestinal tract can be produced quite readily with a number of noxious stimuli such as histamine in bees wax, adrenalin, pitressin, and so forth. Second, individuals who have never had ulcers frequently develop them when they are subjected to the stress of infections, intracranial lesions, burns, or severe trauma. Why this occurs is unknown; however, the work of Selye and others would suggest that it is related to the "general adaptation syndrome."[14]

b. *Genetic proclivity or the constitutional factors:* The early work which attempted to relate peptic ulceration to certain body-types is interesting, however it is of little help in understanding the genesis of the disease. Recently, more emphasis has been placed on the frequent strong family history in these patients, and the increased incidence of ulceration in patients with type O blood. It has also been demonstrated that patients with duodenal ulcers have a greater secretory cell mass of their stomachs than healthy subjects,[7] and that there is a positive correlation between the number of parietal cells of the stomach and the presence of duodenal ulcer.[15] In summing up some of the more current ideas on the constitutional or possible genetic factors, it can be said that duodenal ulcers tend to occur more frequently in individuals with type O blood and a family history of ulceration, these patients have an increased secretory mass and number of parietal cells of their stomachs, and in some, the expected depression of gastric secretion following a duodenal meal is absent.

c. *Psychologic:* Doctor Wolf and others have shown that meaningful situations which are stressful to the individual will cause gastric hyperfunction, decreased pain threshold of the stomach, and hyperemia with increased fragility of the mucosa.[13] Two studies by Mirsky help illustrate the interrelationship of the physiologic, psychologic, and social factors.[16] He feels that the gastric hypersecretion may be a predisposing factor and not an immediate causative factor. He used the serum pepsinogen level as an index of gastric secretion and in the first study he obtained pepsinogen levels on 2,073 draftees. Subjects with values greater than one standard deviation of the mean value of the adult healthy population were considered hypersecretors. Those with values below one standard deviation of the mean were regarded as hyposecretors. In this study, the 16 weeks of army basic training was used as an environmental situation which might prove stressful to some and not to others. He obtained complete gastrointestinal x-rays and psychologic tests on 63 of the hypersecretor group and 57 of the hyposecretor group. This first roentgenologic examination revealed evidence of a healed duodenal ulcer in three men and an active ulcer in one, all from the hypersecretor group. The examinations were repeated on all but thirteen men between the eighth and sixteenth week of training. Evidence of an active duodenal ulcer was found in an additional five individuals whose original x-ray studies were negative. They were also from the hypersecretory group. Therefore, nine of the individuals in the hypersecretory group were found to have or to have developed an ulcer, whereas none of the patients in the hyposecretory group did. In the second study, which is a long-term study and as yet incomplete, serum pepsinogen levels were determined on 1,600 children, 16 years of age or less, and 4,460 adult men and women. The 2% with the highest levels were considered hypersecretors and the 2% with the lowest were considered hyposecretors. During the several years that this study has been conducted, a fairly large number of "healthy" hypersecretors have developed duodenal ulcers while none of the hyposecretors have. In this group, the noxious stimulus occurred spontaneously in their everyday life and, in most cases, it was possible to determine this significant event.

d. *Ecologic:* This is the subject of the talk by Doctor Honska and I shall omit it from my discussion.

Even from this oversimplified and abreviated presentation on the possible etiologies

of peptic ulcer disease, it is apparent that in each case, there are probably many causative and contributing factors. This is important both to the research worker who might measure only one or two parameters and the clinician who has a fixed therapeutic regimen for each patient. We might learn more about this disease and our patients if we ignored the tendency to catalogue all the historical, laboratory, and therapy response data, and studied each patient as a person with certain individual psychologic, physiologic and therapeutic responses. A classification of this type would be more meaningful than the presence or absence of a "little crater."

Lee Honska, M.D.: The duodenal ulcer patient may classically be described as a long, thin, hungry type with intense anxiety, insecurity, resentment, guilt and frustration. The problem of peptic ulcer is wide spread throughout the world. Of interest is the fact that the pattern of the disease varies from place to place and from time to time in incidence and various characteristics. In North England duodenal ulcer is more common than in South England. There is more seasonal variation in ulcer disease in the United States than Brazil. In the 19th Century duodenal ulcers were more common in women and now they are common in men.[17] In discussing the epidemiology of peptic ulcer I will try to point out some of these problems.

Peptic ulcer occurs more frequently in urban than in rural areas. Davies stated that ulcer is unknown in primitive uncivilized races, however, I might add that he forgot to mention that gastroenterologists and x-ray machines are also unknown in these areas. Autopsy figures suggest that 10% of all people suffer from a duodenal or gastric ulcer sometime in their life. Although an ulcer may occur at anytime the highest incidence occurs between 17 and 50 years and Caucasians are affected more than the colored races.

In regard to sex, duodenal ulcer with its complications of perforation and hemorrhage occurs more frequently in males than in females, a ratio of 10 to 1.[18] This is an interesting change since the beginning of the 19th Century when peptic ulcer was more common in women.[19] This change has occurred in Germany, France and all English speaking countries.[20] During the 19th Century a woman's opportunities were limited. Success usually meant getting married and this had to occur between 19 and 30 years or she was a failure. She had to remain sweet and mild and fainting was the only outlet for her emotions and hostilities. With the 20th Century, a women's place in life has changed. If she works and is a failure she always can retreat back to the home without disgrace. Men, on the other hand, have had no change in their responsibility and frequently have lost the emotional support from the wife at home as well as having increasing competition with her at the office. Thus, it may be that there is not a true increase in the number of peptic ulcers in men but a relative decrease in those of women.

Genetic factors probably play some role in peptic ulcer disease. There are numerous examples of several cases occurring in the same family and to support the genetic theories is the fact that the people with group O blood are 35% more likely to have peptic ulcer than patients with other blood groups.[21] Doctor Welsh has mentioned other evidence supporting the hypothesis that there is a genetic aspect of the disease.

Environment has been discussed in relation to peptic ulcers. In Brazil where the temperature is more constant during the year, we do not see the seasonal variations reported in this country.[17]

Boles and Westerman[22] have correlated temperature changes with hemorrhage from duodenal ulcer and showed that this occurs more often when the temperature changes are the greatest; November, December, March and April. We also know that more people perforate their ulcer on Friday and Saturday and after 4 p.m.

In regard to habits, smoking has been incriminated, but the only thing that is known is that more ulcer patients smoke, and smoke more than the general population and that this makes very little difference in the healing rate of the ulcer.[23]

The socio-economic factors of peptic ulcer are interesting and evidence may be produced to prove any question. Sarah Jordan reports a higher incidence in the higher economic class and Doctor Jones reports a higher incidence in the lower economic class. It would appear that this difference depends on the type of patient they see.

Occupation has been extensively studied in patients with peptic ulcer. Porter[24] has reported some interesting findings in his work with monkeys. One monkey (his executive monkey) was given the responsibility of pushing a bar to prevent a control monkey and himself from receiving a shock. The executive monkey developed an ulcer, the control monkey did not. His conclusion from this was that it is "not the amount of work that is involved but the amount of responsibility," or transposing this to humans, the amount of responsibility the patient feels.

Certain other diseases have been related to peptic ulcer. Emphysema, cirrhosis and coronary disease[25] each show a relatively high correlation with the presence of duodenal ulcer. But similarly almost every other disease has also been associated in some degree with peptic ulcer by someone.

At the recent World Congress of Gastroenterology, experts from all over the world discussed the epidemiology of peptic ulcer. With understandable patriotism, the Indian absolved spices; the Frenchman, wine; the Brazilian, coffee. Others made positive correlations and stated that ulcers reappeared with the seasons, with the resumption of smoking, with overeating, with undereating, with making too much money, with having too little money, and naturally with emotional stress.

CLOSING REMARKS by Stewart Wolf, M.D.

We are very much indebted to Doctors Welsh and Honska for their scholarly exposition of the many aspects of peptic ulcer disease. They have made it clear that there is still a great deal to learn about gastric secretion and the vulnerability of the duodenal mucosa. They have both implied to us, and I think their implication is attractive, that in searching for the cause of a disease we search for the will-o'-the-wisp. It would appear that there are many circumstances which enter the picture and ultimately determine sickness or health. Among these are the inherent characteristics of the person, his way of life, and the multitude of hazards that he may encounter along the road. As soon as we give up the idea of a single cause and concern ourselves with relevant factors, our understanding of peptic ulcer, and indeed of other disease processes, will be enhanced.

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ABSTRACTS

X-Ray Studies in Establishing Identity and Manner of Death

ERNEST LACHMAN, M.D.*

The Journal of Criminal Law, Criminology and Police Science, Vol. 50, No. 2, July-August, 1959

X-rays are an indispensable tool of the crime detection laboratory. The roentgenographic method permits us to verify or exclude the human origin of parts submitted and to determine the approximate age of a cadaver, particularly under 25, without destruction of its soft tissues. Roentgenographic study of the fetal skeleton allows the determination of a viable age and of full term, roentgenograms of the adolescent hand can predict menarche within a range of six months. After 25 years of age, closing of cranial sutures, presence or absence of teeth, progressive calcifications in the costal cartilages and secondary changes in the joints serve as less reliable age indicators.

Identification of the individual x-rays is based on skeletal features typical for the individual as established by previous roentgenograms, such as size and shape of the paranasal sinuses, congenital deformities, old fractures, curvatures of the spine, residues of previous illnesses and minor features of individual bones.

As to determination of manner of death, the demonstration of vital fractures, and presence and direction of bullets including their point of entrance are within the province of roentgenography. Numerous examples from the author's experience and the literature illustrate the discussion.

*Professor and Chairman of the Department of Anatomy.

Relative Potencies of Chlorpropamide And Tolbutamide in Man

KELLY M. WEST* and STANLEY R. McCAMPBELL**
Annals of The New York Academy of Sciences 74: 473-477, March 1959

Chlorpropamide, 1 gm., produced significantly greater hypoglycemic responses in normal subject one hour after ingestion than did tolbutamide, 1 gm. Chlorpropamide, 0.25 gm. and 0.5 gm., produced hypoglycemic responses in normal subjects two hours after ingestion comparable to doses of tolbutamide twice as great. Since is it unlikely that either drug had been appreciably metabolized or excreted one to two hours after ingestion, the relatively greater potency of chlorpropamide probably is not attributable entirely to its longer biological half time.

Seven normal subjects had serum chlorpropamide levels averaging 11.5 mg. per cent two hours after ingesting chlorpropamide, 1 gm., indicating that about one-third of this dose was present in the plasma at that time.

Chlorpropamide, 0.5 gm., significantly increased the rate of disappearance of intravenously injected glucose. In one subject the effect of an intravenous injection of chlorpropamide, 0.5 mg., on the glucose disappearance rate was strikingly similar to that of 1.3 units of insulin administered intravenously.

Two findings that have resulted from studies carried out since this report was drafted are of considerable interest in connection with the initial findings. We found that serum levels of chlorpropamide two hours after the drug was administered orally were much higher than serum levels of tolbutamide after comparable doses of that drug. We found also that when chlorpropamide and tolbutamide were administered intravenously the hypoglycemic effects during the first hour were not significantly different.

*Assistant Professor of Medicine.
**Instructor in Medicine and in Preventive Medicine and Public Health.

The Comparative Pharmacology of Tolbutamide, Carbutamide, Chlorpropamide And Metahexamide in Man

KELLY M. WEST* and PHILLIP C. JOHNSON**
Metabolism, 8: 596-605, July 1959

The absorption of orally administered chlorpropamide and metahexamide in man is rapid and is usually virtually complete in two to three hours. The absorption of carbutamide is somewhat slower, and the absorption of tolbutamide occurs much more slowly. Absorption of each of these four drugs is eventually virtually complete.

We found no evidence of differences in the potencies of tolbutamide, metahexamide and chlorpropamide in acute experiments in which serum levels of comparable magnitude were produced after oral administration. However, hypoglycemic responses of normal subjects to metahexamide immediately after intravenous administration suggested that it was about three to four times as potent as tolbutamide.

The serum levels of tolbutamide, metahexamide, carbutamide and chlorpropamide after intravenous injection are much too high to be compatible with the concept that they are uniformly distributed in the extracellular fluids. Direct measurements of tolbutamide, chlorpropamide and carbutamide in extracellular fluids other than serum such as edema, ascitic and pleural fluid indicate that levels in these extracellular fluids are considerably lower than in serum.

The serum levels of tolbutamide, chlorpropamide and carbutamide 40 minutes after an intravenous injection

are virtually identical, suggesting that there are no substantial differences among these three compounds in respect to the volume in which they are distributed in the body.

*Assistant Professor of Medicine.
**Associate Professor of Medicine.

The Intravenous Glucose Tolerance Test

KELLY M. WEST* and DOROTHY ANTONIA WOOD**

The American Journal of the Medical Sciences **238**: 25-37, July 1959

When a glucose load of a standard size is administered rapidly intravenously, the rate of fall of the blood glucose during the first 25 minutes after the injection may be influenced by several factors which are not related to the rate of glucose utilization. Some of these factors are the size of the glucose space (which is in turn related to body size), the height of the blood glucose before the injection, the renal excretion for glucose, and the size of the glucose load.

In normal subjects, the rate of disappearance of the blood glucose after a rapidly administered glucose load of 25 gm. usually remains exponential in regard to time during the period 25 to 60 minutes after the injection at a time when the blood glucose is falling from levels near the renal threshold for glucose to levels at or just above preinjection levels. It seems likely that during this period the rate of disappearance of the absolute glucose is well related to glucose utilization although some other factors are probably operative. On the other hand during this same period the rate of disappearance of the increment blood glucose (or glucose excess) is inconstant in that the log plots of these values against time form a convex rather than a straight line. Evidence is presented which suggests that the advantages of analyzing increment rather than absolute values which have been described previously were apparent rather than real.

Glucose tolerance can be measured by administering a load rapidly intravenously of sufficient size to produce the hyperglycemia of a "physiologic" magnitude and by determining the rate of fall from a time the blood glucose is near the renal threshold for glucose until it is approaching the preinjection level. Evidence is presented suggesting that under certain conditions, fairly accurate estimation of glucose tolerance may require only one or two determinations of blood glucose.

*Assistant Professor of Medicine.
**Medical Technologist, Experimental Therapeutic Unit.

The Tuberculin Test

HARRIS D. RILEY, Jr.*

American Practitioner and Digest of Treatment **10**: 1487-1488, September 1959

*Professor of Pediatrics.

Expeditious Evaluation of Circumscribed Pulmonary Shadows

ALLEN E. GREER,* JOHN M. CAREY** and NAZIH ZUHDI***

The Journal of the AMA **171**: 1783-1786, November 1959

Of 85 patients with asymptomatic pulmonary rounded opacities one-half had neoplasms and over one-third had primary carcinomas of the lung. Since the most important consideration is whether these lesions are benign or malignant, early exploration is recommended.

*Assistant Professor of Surgery.
**Instructor in Surgery.
***Resident in Thoracic Surgery.

Treatment of Diabetes Mellitus With Orally Administered Compounds

KELLY M. WEST* and STANLEY R. McCAMPBELL**

Southern Medical Journal, Journal of the Southern Medical Association **52**: 655-658, June 1959

*Assistant Professor of Medicine.
**Instructor in Medicine and in Preventive Medicine and Public Health.

Appointments Announced By Board of Regents

John W. Kelly, Ph.D., former member of the Medical College of Virginia, has been appointed an associate professor of pathology at the University of Oklahoma School of Medicine.

Teaching in the anatomy department of the Virginia college from 1951 to 1958, Doctor Kelly spent one year as a guest investigator at the Institute for Cell Research, Medical Nobel Institute, Stockholm, Sweden. He was a John Simon Guggenheim fellow.

Doctor Kelly received his Ph.D. from the University of Pennsylvania. He is studying the histochemistry of mucopolysaccharides and of anionic disease dyes.

University regents also have approved appointment of the following to teach on a part-time basis: Harry Martin, Ph.D., staff clinical psychologist at the Oklahoma City Veterans Hospital, instructor in psychiatry; Galen P. Robbins, M.D., former assistant in medicine and fellow in cardiology at Baylor University College of Medicine, Houston, clinical assistant in medicine; Walter Robert Cooper Jr., D.D.S., periodondist formerly of Tulsa, clinical assistant in oral surgery.

PRESIDENT'S LETTER

No More Lincolns

Would one of history's most revered citizens have risen from a humble beginning to a position of adulated stature if he had lived in today's welfare environment?

With this challenging thought in mind, Abraham Lincoln's birthday takes on more significance than its usual role of routine public acceptance as just another national holiday, or, in many cases, simply another day of relief from productivity.

It seems fair to assume that a mid-twentieth century Abe would have been deterred, if not completely thwarted, in his efforts to shake off the dust of illiteracy, poverty and obscurity. The offspring of an impoverished itinerant farmer has little opportunity today to even recognize the values of personal initiative and enterprise, much less have the old fashioned gumption to strive for such qualities.

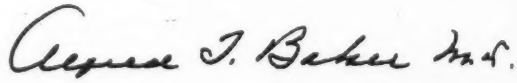
A combination of welfare checks, subsidies and land banks would cast a paternalistic shadow of inertia over such unprofitable and ignominious pastimes as splitting rails. And would Abe have read his books by firelight, or would he have sat idle and complained that the REA was denying him his birthright of electric power?

Today's regimented education, which places memory power above thinking power, would probably not have challenged Lincoln's hungry mind, nor have produced an individualistic thinker of his caliber.

Would he try to better himself and the land in which he lived, or would he have turned his considerable talents toward becoming a vociferous drone who expended his energy to achieve an increasingly larger dole from the public udder?

Lincoln's trademark is that of an uncouth, backwoods lad who came to be the savior of the Union and the symbol of pioneer spirit and American Democracy. Let's not relegate his spirit to a vaguely-associated national holiday, but employ his memory as a daily yardstick to measure the philosophical course of our government and our way of life!

What would our country be today without Honest Abe . . . what will the future hold with no more Lincolns?



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SEARLE

Special Articles

Factors Relating To Physician Distribution in Oklahoma 1950-1959

Part 1 of a Series

ROBERT C. LOWE, M.D.*
JAMES A. HAGANS, M.D.*
PEARL D. FISHER, Ph.D.*

THE CURRENT STUDIES of the health status and the medical care of the older age groups in the population cannot be accomplished without reference to the medical care resources available to the whole population. Of these resources, a competent and cohesive practicing physician population is necessary for adequate development, operation and function of all health facilities and personnel. It is essential that the medical profession prosper as its standards of service progress and the scope of its services broadens. This is necessary in order to attract others into the profession that it may reproduce itself and its quality at whatever rate is required by the demands for its services.

In what way do hospitals and city populations affect the location of physicians?

The trend of loss of physicians from the rural and smaller urban communities in Oklahoma described by Belcher[1] has continued. Associated with these net losses there has been a redistribution, the pattern of which is no doubt being infuenced by a number of factors. The extension of the quality and scope of service of which the profession is capable is logically a major influence. Population redistribution, supply and demand, community or regional economics, the geographic availability of hospital service of increasing quality and required complexity are some of the secondary and related forces currently at work to make the most effective use of the resources available.

Table I indicates that the net loss of physicians is inverse to community size. Between January 1, 1950 and January 1, 1959 only those communities with populations over 20,000** (1950) have, as a group, shown a net increase. On the other hand, of 204 communities with populations under 2500 (1950), 37 had no doctor of medicine in 1950. By 1959, 7 of these had acquired physicians but 53 more had lost theirs, (1-4) an increase to 83 (40.7% of the original group) without doctors of medicine. The evidence indicates that the end of this trend has not been reached.

The trouble with such statistical data, lacking information on offsetting trends and the rest of the picture, is that they may be interpreted offhand as deterioration in medical care. Such an interpretation does not consider the extent to which these changes are an expression of improvement brought

*All three of the authors are with the Department of Preventive Medicine and Public Health, University of Oklahoma School of Medicine; Doctors Hagans and Fisher are also with the Biostatistical Unit of the Department of P.M. and P.H.; in addition, Doctors Lowe and Hagans serve with the Department of Medicine.

**Excluding communities in Tulsa, Oklahoma and Muskogee counties.

about by other factors in the redistribution. It is evident that further information is needed. Table II shows the relationship of a hospital facility and community size to the changing pattern in communities with populations between 750 and 3500. Section A indicates that essentially the same proportion of physicians remained from 1950 to 1959 regardless of community size or the presence or absence of a hospital facility. It is worth noting, however, the tendency for communities with hospital facilities to retain fewer when the community size is small (i.e. less than 1500) than when it is larger (i.e. from 2500 to 3500).

Section B (table II) indicates that in small and intermediate size communities with hospitals a significantly greater proportion of new physicians was present in 1959 than was the case for the same size communities without hospitals. It should be noted that the proportion of new physicians in communities with hospitals decreased as the size of the community increased. The in-

(Continued on Page 113)

TABLE I

Pop. Range	No. of Towns	M.D.s 1950	M.D.s Remaining	M.D.s New	M.D.s 1959	% Change '50-'59
<750	106	97	48	11	59	-39.2
750-1499	61	94	39	24	63	-33.3
1500-2499	37	86	40	27	67	-22.1
2500-3499	20	86	47	24	71	-17.4
3500-4999	10	63	40	23	63	0.0
5000-9999	20	201	110	55	165	-17.9
10,000-14,999	6	80	53	17	70	-12.5
15,000-19,999	7	184	111	67	175	-4.9
20,000-36,000	6	163	115	71	189	+15.9

TABLE II

SECTION A: % of M.D.s present in 1950 and remaining in 1959—(chi square analysis*)			
Pop. Range	without hospitals	with hospitals	statistical significance of difference
750-1499	(33/74) 45%	(6/20) 30%	p > .05
1500-2499	(23/49) 47%	(17/27) 46%	p > .05
2500-3499	(9/20) 45%	(38/66) 58%	p > .05
Significance of Difference (vertically)	p > .05	p > .05	
SECTION B: % of total M.D.s in 1959 who were new since 1950—(chi square analysis*)			
750-1499	(13/46) 28%	(11/17) 65%	p < .01**
1500-2499	(8/31) 26%	(19/36) 53%	p < .02**
2500-3499	(7/16) 44%	(17/55) 31%	p > .05
Significance of Difference (vertically)	p > .05	p < .02**	
SECTION C: % change in total physicians 1950 to 1959—(chi square analysis*)			
750-1499	(28/74) -37.8%	(3/20) -15.0%	p < .05**
1500-2499	(18/49) -36.7%	(1/37) - 2.7%	p < .02**
2500-3499	(4/20) -20.0%	(11/66) -16.6%	p > .05
Significance of Difference (vertically)	p > .05	p > .05	

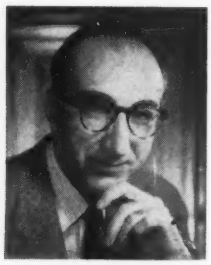
*p represents the probability that a difference as great or greater than that observed would have occurred by chance alone; when the value was .05 or less (1 in 20 or less) it was regarded as sufficiently rare as a chance occurrence to indicate a significant difference.

**indicates statistically significant difference.

News

TOP TALENT FEATURED FOR ANNUAL MEETING

ALLEN



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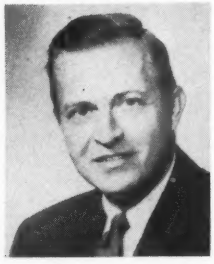


SKIBBINS

SOFIELD

VIRTUE

With twelve distinguished guest speakers confirmed, nearly one hundred high-quality scientific and technical exhibits scheduled, and an outstanding social program in the making, the Oklahoma State Medical Association's 1960 Annual Meeting promises to attract a record turnout. Oklahoma City will serve as host city this year, with all scientific events concentrated in the Municipal Auditorium and all social activities arranged at the Skirvin Tower Hotel. Dates for the Annual Meeting are May 2, 3, 4, 1960.

Under the leadership of General Chairman C. M. Bielstein, M.D., and Scientific Works Chairman Bert F. Keltz, M.D., the program has been completed at an early date. The unique composition of the Scientific Works Committee has contributed greatly toward the achievement of a diversified program of general interest. This year, the majority of specialty societies were represented by their respective presidents, as was the Oklahoma Chapter of the American Academy of General Practice.

Speakers Named

Listed as guest speakers for the event are: Edgar V. Allen, M.D., Internal Medicine, Rochester, Minnesota; Grayson Carroll, M.D., Urology, St. Louis, Missouri; Michael DeBakey, M.D., Surgery, Houston, Texas; Vincent J. Derbes, M.D., Dermatology, New Orleans, Louisiana; Eugene L. Derlacki, M.D., E.E.N.T., Chicago, Illinois; Murdina Desmond, M.D., Pediatrics, Houston, Texas; Benjamin Felson, M.D., Radiology, Cincinnati, Ohio; Schuyler Kohl, M.D., Obstetrics and Gynecology, Brooklyn, New York; Harold Rosen, M.D., Psychiatry, Baltimore, Maryland; Mr. Gerald J. Skibbins, Public Opinion, Princeton, New Jersey; Harold A. Sofield, M.D., Orthopedic Surgery, Oak Park, Illinois; and Robert W. Virtue, M.D., Anesthesiology, Denver, Colorado.

Public Opinion Expert Featured

Mr. Skibbins, Research Executive for the Opinion Research Corporation of Princeton, New Jersey, will provide an interesting di-

version from the scientific program. He will be the featured speaker at Tuesday's luncheon, where he will discuss public opinion surveys which his firm has conducted on behalf of the medical profession.

Panels Planned

The scientific program will be highlighted by two symposia-panel disucssion features.

On Tuesday morning, May 3, Doctors DeBakey, Allen and Virtue will present papers concerning current concepts of vascular disease. These formal presentations will be followed by a panel discussion and a question and answer period involving the three guest lecturers and a local pathologist.

The next morning, May 4, will find the entire concluding portion of the annual meeting devoted to rehabilitation, with particular emphasis on hemiplegia, spinal cord injuries, amputation and arthritis. This segment of the program will be under the direction of the Institute of Physical Medicine and Rehabilitation of NYU's Bellevue Medical Center. Local physical medicine personnel will cooperate in the presentation and will furnish demonstration subjects and equipment.

Social Highlights

Scientific activities will be adequately blended with social events in an effort to offer attenders a typically well-balanced meeting. Entertainment Committee members are now negotiating for a big-name orchestra to entertain at the President's Inaugural Dinner-Dance, which will be held in the Persian Room of the Skirvin Tower Hotel on Tuesday evening, May 3. The program will follow the usual pattern of dinner, installation ceremonies and dance.

President Alfred T. Baker, M.D., Durant, will culminate a busy year as the association's top executive officer when he presents the President's gavel to Walter E. Brown, M.D., Tulsa.

Entertainment Extra

Physicians and their wives who attend the meeting will be able to take advantage of additional topflight entertainment in the form of the Broadway production of "Look Homeward, Angel," which is scheduled at the Warner Theater on May 4 and 5. The Pulitzer Prize Winner, starring Miriam Hopkins, will be brought to Oklahoma City through the sponsorship of the Junior League.

Approximately two hundred tickets remain for each performance in price ranges of $5.75, $5.15, $3.75 (balcony) and $2.75 (balcony). The compactness of the theater should minimize the disadvantage of a rather limited selection of seats at this time.

Ticket orders should be accompanied by check and mailed to the Winn Ticket Agency, 135 West Main, Oklahoma City.

Oklahomans Participate In Texas Law-Science Week

Nine Oklahomans participated in Law-Science Week, held February 15-20 in Houston, Texas. Physician participants were: Earl D. McBride, M.D., William L. Waldrop, M.D., Louis J. West, M.D., and Robert A. Schneider, M.D., all of Oklahoma City.

The legal profession of the state was represented by Robert D. Looney, Fred M. Mock, and O. A. Cargill, Jr., of Oklahoma City, and Bryan W. Tabor, Tulsa. Mr. Ralph Snyder of Oklahoma City was also present.

Law-Science Week was organized by Hubert W. Smith, LL.B., M.D., Professor of Law and Legal Medicine, University of Texas, Director of the Institute and Chancellor of the Law-Science Academy. He was assisted by Warren P. McKenney, LL.B., Executive Assistant of the Institute and the Academy.

The six day program encompassed two short courses, directed to lawyers, physicians and claims men who are interested in medico-legal problems. The first short course, sponsored by the Law-Science Academy and Foundation was held February 15-17. The second, sponsored by the Law-Science Institute, was held February 17-20. Subject matter for both courses involved personal injury problems and medico-legal trial technique.

"Medicare" Expands Scope of Care

The Dependent's Medical Care Program has restored many of the benefits eliminated from the original program during the budgetary cutback of 1958. In October 1958, decreased appropriations rendered the Medicare Program financially unable to meet its obligations as originally established and agreed upon by state medical societies and the Department of Defense.

Flashback

Oklahoma had just completed re-negotiation of the contract for another fiscal year when the surprise announcement of the cutback was made at a special meeting in Washington, D.C. The abrupt about-face of the Federal government was followed, in turn, by a special meeting of the OSMA's House of Delegates, which promptly withdrew the association from any official sponsorship of the program and terminated all contractual arrangements. Blue Shield retained its position as fiscal agent on a month-to-month basis. Very basically, the restricted program slightly curtailed obstetrical benefits, eliminated all but emergency surgery and required military dependents who resided with their sponsors to first report to a military hospital for care. If facilities and/or personnel were found to be unavailable, the prospective patient would be issued a permit authorizing civilian care.

New Program Outlined

While the new program liberalizes the benefits to some extent, it is not a complete restoration of the original agreement.

Probably the most important *unchanged* feature of the program is the continued requirement of the permit system. The law states that spouses and children residing with their military sponsor are not eligible for civilian medical care and hospitalization privileges under Medicare *unless* the military medical facility nearest them is inadequate or unavailable. In such a case, a permit shall be issued by the Military Commander to qualify the eligible dependent to receive *authorized* civilian care at Federal expense. This permit must accompany the physician's claim, except in physician-certified emergencies. The details of this procedure remain unchanged and may be found on page two of the Medicare Manual.

The following types of care have been *restored* to authorized coverage:

1. Certain Surgery.
2. Acute emotional disorders of an emergency nature.
3. Laboratory and X-ray procedures preceding or following hospitalization *and* surgery.
4. Treatment of injuries on an out-patient basis.

Not restored, however, are neonatal visits and termination visits.

Surgery

Among the surgical procedures restored, are Stapes mobilization, eye surgery for glaucoma, cataracts and strabismus, and surgery for skeletal defects such as club foot. Payment for these procedures and the many others re-activated under the expanded program will be authorized only if the physician certifies that the surgery is for functional improvement or restoration.

Acute Emotional Disorders

Acute emotional disorders are now authorized and compensable if the condition constitutes a threat to the life or health of the patient. Twenty-one days coverage is the maximum allowed unless approved by prior arrangement.

Laboratory and X-Ray

Pre- and post-hospitalization tests and procedures, as authorized under the expanded program, do not include payment for outpatient office visits or physical examinations. They do include such tests and procedures when performed in or out of the hospital if the resulting surgery and hospitalization are authorized. Up to $75.00 is allowed pre-operatively and up to $50.00 post-operatively.

Out-Patient Treatment of Injuries

Treatment of bodily injuries on an outpatient basis is now authorized, but is lim-

ited to treatment of fractures, dislocations, lacerations and wounds. Compensation for such services will include payment for diagnostic and therapeutic tests and procedures as authorized by the physician.

The patient is still responsible for payment of the first $15.00 of the physician's total charges. Payment for laboratory tests, pathological and radiological examinations must not exceed $75.00.

Government Will Control and Interpret

Voluminous details concerning the expanded program will be interpreted by the Medicare Office of the Department of Defense. Neither the OSMA nor Blue Shield have jurisdiction in this area, although the latter will process claims as the fiscal agent.

Keogh Legislation Faces Crisis

The long-awaited Smathers-Morton-Keogh-Simpson Bill is now facing its most most crucial test in several sessions of Congress. It is now before the Senate Finance Committee in a make-it-or-break-it culmination of nine years' promotion by the medical profession and other self-employed groups.

Last year, the House of Representatives passed the measure and referred it to the Senate where it rested without action until congressional adjournment. Thus, the recommendation of the Senate Finance Committee will have great influence on its final disposition.

OSMA Action

Under the direction of J. R. Stacy, M.D., the association's Federal Legislative Committee has been conducting a write-in campaign among the membership, as have other interested associations and groups. The letters are being directed to Senator Robert S. Kerr, Senate Office Building, Washington, D.C. Senator Kerr is the second-ranking member of the Finance Committee and will undoubtedly have much to say about the committee's recommendation. Although the senator is aware of the medical profession's general attitude toward this legislation, a display of unity at this time would assist him in properly representing the self-employed of his state. County medical societies and individual physicians are encouraged to write.

Tax Equity for the Self-Employed

The purpose of the bill is to permit a self-employed person to defer income tax each year on $2500 or 10% of income, whichever is less, but not to exceed $50,000 during a life-time. For those who have reached the age of fifty prior to the effective date, provision is made to allow additional amounts to be set aside in order to build up an adequate fund during productive years.

Under the bill, a person may claim a deduction for an investment in (1) a restricted retirement policy (life, endowment or annuity; but deductions are not allowed for costs allowable to the life insurance protection); or, (2) a restricted retirement fund trusteed with a bank.

Why Is It Necessary?

High taxes and inflated living costs make it difficult for the self-employed person to set aside money for retirement, in the absence of tax deferments which are already available to corporation employees. In many instances, qualified young men are going on a payroll rather than striking out for themselves as enterpreneurs. This legislation would help counteract this shift away from individual enterprise.

About ten million persons are now penalized by the tax laws. The Old-Age and Survivors Insurance program is intended to provide the minimum benefits necessary for a subsistence standard of living, and self-employed persons should have the right, as do salaried employees, of providing a second layer of income for retirement and survivorship's benefits. This is even more desirable and equitable in the case of physicians, who have elected to abstain from participation in the Social Security scheme.

If the bill passes, physicians will not pay taxes on the set-aside until it is withdrawn from the selected fund, thus paying a lesser tax rate during less-productive years.

DAVID M. HUME, M.D.

Third Oklahoma Colloquy Planned

Use of the adrenal steroids in surgical cases, in collagen disease and in allergic problems will be among the clinical aspects covered by guest lecturers at the Third Oklahoma Colloquy on Advances in Medicine March 24-26 at the University of Oklahoma Medical Center.

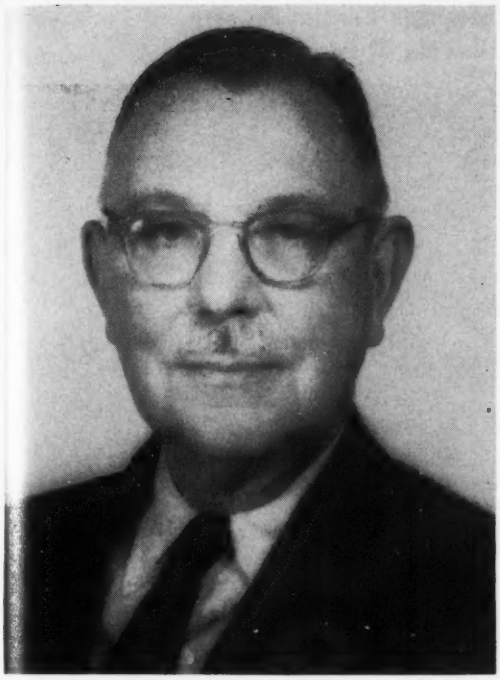
David M. Hume, M.D., chairman, Department of Surgery, Medical College of Virginia, Richmond, will discuss use of the steroids in the surgical patient at a sectional dinner meeting sponsored by the Oklahoma City Surgical Society March 25 at Glen's Hick'ry Inn.

In addition, Doctor Hume will address a general session March 24 on "The Mechanisms Controlling Adrenal Cortical Secretion in Response to Trauma." Doctor Hume has published extensively in the areas of adrenal cortical secretion and of renal and other tissue transplantation.

Bram Rose, M.D., Ph.D., associate professor of medicine, McGill University, will appear before both morning and afternoon general sessions March 26. He will talk on the use of anti-inflammatory steroids in the control of collagen diseases and in the management of allergy.

President-elect of the American Academy of Allergy, Doctor Rose is on the editorial boards of the Journal of Allergy and the International Archives of Allergy and Immunology. His most recent research has

BRAM ROSE, M.D., Ph.D



LAWSON WILKINS, M.D.

been carried out on antigen-antibody mechanisms with particular reference to those related to the common forms of allergy.

Lectures by Lawson Wilkins, M.D., professor of pediatrics, Johns Hopkins University School of Medicine, are scheduled at the morning session March 25 and at an afternoon section meeting sponsored by the Oklahoma City Pediatrics Society the same day.

Doctor Wilkins' studies have helped clarify the problems of abnormal sex differentiation and gonadal dysgenesis. He is author of "The Diagnosis and Treatment of Endocrine Disorders in Childhood and Adolescence."

His general session lecture is entitled "Diagnosis and Treatment of the Adrenogenital Syndrome." At the Pediatrics session, he will speak on "Abnormalities of Sex Differentiation: Etiology, Diagnosis, Selection of Sex and Treatment."

The three are among 15 guest speakers and 13 members of the Medical Center faculty who will present papers during the conference on adrenal steroids. Special interest meetings also are planned in the fields of medicine and basic science.

Oklahomans Attend Blue Shield Conference

More than three hundred delegates, including state and local medical society officers and secretaries, as well as Blue Shield Plan physician-trustees and executives, attended the 1960 Blue Shield Professional Relations Conference which was held February 1-3 at the Drake Hotel in Chicago. President A. T. Baker, M.D., and Prepaid Medical Care Committee members, Carl Lindstrom, M.D., and R. G. Obermiller, M.D., represented Oklahoma.

The Professional Relations Conference program was prepared under the direction of the National Association's Professional Relations Committee and had as its theme this year, "Facing the Facts—In the Future of Blue Shield."

"The new year presents a serious challenge to Blue Shield and to the entire voluntary health care movement," the advance publicity stated, "for the simple reason that it is an election year and we can expect a supreme effort by the backers of Forand-type legislation to get their program passed in Congress. To give our physician delegates as much of an up-to-date picture as possible, we have scheduled discussions on federal legislative activities and on Blue Shield activities with regard to the development of programs for senior citizens.

Among the speakers were Doctor John Reckless of Duke University, who discussed "The Facts of Life in the Future of American Medicine;" Doctor Charles G. Hayden, Executive Director of the Massachusetts Blue Shield Plan, who spoke on "Evolving New Benefit Patterns to Meet the Demands for Expanded Forms of Coverage—An Examination of Factors of Interest and Importance to the Profession;" and Doctor Ira C. Layton, of Kansas City, Missouri, who talked about "The Dynamics of Blue Shield and Their Relation to the Future of Private Medical Care." Also on the program were Thomas M. Newell, Director, National Field Service, New York City, whose subject was "Public Opinion and Its Application in Shaping Future Developments in Blue Shield," and Doctor Donald Stubbs, Chairman of the

Board of the National Association of Blue Shield Plans, who talked about "The Plan Governing Board—An Examination of Concepts Affecting Its Composition Functions and Responsibilities."

AAGP Will Hold 12th Annual Scientific Assembly

The 12th Annual Scientific Assembly of the American Academy of General Practice will be held in Philadelphia's Convention Hall, March 21-24, 1960. Approximately 4,000 family doctors and 3,000 residents, interns, exhibitors and wives will attend the four-day program which will highlight recent progress in medicine and surgery.

The Philadelphia Assembly program lists 31 prominent medical educators who will discuss a variety of subjects ranging from arthritis and anemia to surgery, geriatrics and mental health. The doctors will also visit more than 100 scientific and 300 technical exhibits.

During the meeting, the doctors will have a chance to be on the other end of a stethoscope during physical examination sessions. More than 600 doctors took physical examinations at the last Academy meeting. Many learned, for the first time, that they needed immediate medical attention.

Fifty Year Club to Be Organized Within AMA

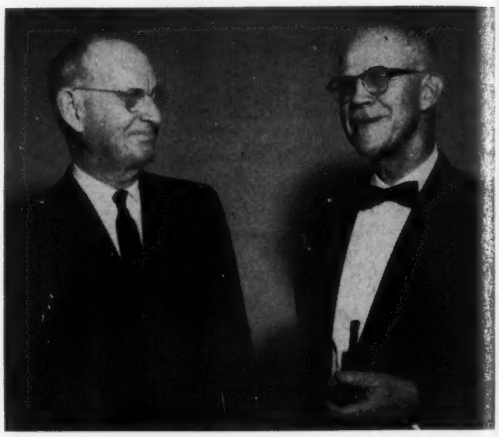
J. H. McCurry, M.D., of Cash, Arkansas, advises that he has the approval of the American Medical Association to organize a Fifty Year Club within the AMA. Doctor McCurry is anxious to hear from physicians who have been in practice fifty years or more who desire to become members of this club, giving their name and a complete address.

The first meeting is to be held in Washington, D.C. at the Clinical meeting November 29 to December 2, 1960.

OB-GYN Exams Scheduled

The American Board of Obstetrics and Gynecology has scheduled examinations (Part II), oral and clinical for all candidates from April 11 through 16, 1960 at the Edgewater Beach Hotel, Chicago, Illinois.

Medical Center Presents Service Awards



W. K. West, M.D., left, and Dean Mark R. Everett at annual service award ceremonies at the University of Oklahoma Medical Center. Doctor West received a 40-year pin.

Sixty-one faculty members, including 11 preceptors, were cited at the University of Oklahoma Medical Center's fourth annual service awards program December 10.

Heading the list was W. K. West, M.D., professor of orthopedic surgery, honored for serving 40 years on the School of Medicine faculty.

Pins were awarded by Mark R. Everett, Ph.D., dean and director of the Center, who was presented a 35-year citation by Lloyd E. Swearingen, Ph.D., vice-president in charge of research and development at the University of Oklahoma, Norman.

Other 35-year pins went to: James B. Eskridge Jr., M.D., professor of obstetrics; Leroy Long, M.D., clinical professor of surgery; Joseph C. MacDonald, M.D., professor of otorhinolaryngology; Leo Starry, M.D., professor of surgery.

Thirty year awards were given: Louis H. Charney, M.D., associate professor of medicine; Ephraim Goldfain, M.D., assistant professor of medicine; R. Q. Goodwin, M.D., professor of medicine; W. Jeanne Green, Ph.D., assistant professor of pathology; Joseph W. Kelso, M.D., professor of gynecology; Lawrence C. McHenry, M.D., pro-

fessor of otorhinolaryngology; James F. Moorman, M.D., associate professor of medicine; Charles R. Rountree, M.D., clinical professor of orthopedic surgery; James B. Snow, M.D., professor of pediatrics.

Ernest Lachman, Ph.D., professor and chairman of the Department of Anatomy, received a 25-year pin.

Those who reached the 20-year milestone were: Meredith M. Appleton, M.D., associate professor of urology; Robert B. Howard, M.D., clinical professor of surgery; Harry L. Deupree, M.D., associate professor of obstetrics; Grady F. Mathews, M.D., professor of preventive medicine and public health; Newman S. Matthews, M.D., assistant professor of medicine; Charles M. O'Leary, M.D., clinical professor of surgery; Moorman P. Prosser, M.D., clinical professor of psychiatry; Charles D. Tool, M.D., assistant professor of pathology; Robert I. Trent, M.D., clinical professor of ophthalmology.

Other awards to members of the faculty in Oklahoma City:

Fifteen years—N. F. Barkett, M.D., associate professor of surgery; Robert H. Bayley, M.D., professor of medicine; Florene C. Kelly, Ph.D., professor of microbiology.

Ten years—James C. Amspacher, M.D., assistant professor of orthopedic surgery; C. Alton Brown, M.D., associate professor of medicine; James R. Colvert, M.D., assistant professor of medicine; Sterling T. Crawford, M.D., assistant professor of gynecology; John J. Donnell, M.D., associate professor of medicine; Louis S. Frank, M.D., assistant professor of pediatrics; Walter S. Hendren, M.D., assistant professor of medicine; Robert G. Hirschi, M.D., assistant professor of oral surgery; Ella M. George, M.D., assoriate professor of physical medicine; George H. Guthrey, M.D.. associate professor of psychiatry; Robert P. Holt, M.D., assistant professor of orthopedic surgery; Dick H. Huff, M.D., assistant professor of medicine; Walter Joel, M.D., professor of pathology; Robert W. Kahn, M.D., assistant professor of medicine; A. C. Lisle, M.D., assistant professor of surgery; William G. McCreight, M.D., assistant professor of dermatology; Robert McKee, M.D., assistant professor of obstetrics; Milton J. Serwer, M.D., professor of obstetrics; Harold Sleeper. M.D., assistant professor of psychiatry; Carl T. Steen, M.D., innstructor in psychiatry; L. Duncan Thelkeld, M.D., associate professor of obstetrics; William L. Waldrop, M.D., assistant professor of orthopedic surgery; George L. Winn, M.D., assistant professor of medicine.

The Medical Center this year started awarding service pins to preceptors who have served five or ten years. First awards were made to the following:

Ten years—Carl Bailey, M.D., Stroud; W. C. McCurdy, M.D., Purcell; E. A. McGrew, M.D., Beaver; Joe L. Duer, M.D., Woodward.

Five years—Lynn C. Barnes, M.D., Nowata; Walter H. Dersch Jr., Shattuck; George Gathers, M.D., Stillwater; Robert B. Gibson, M.D., Ponca City; Kenneth Godfrey, M.D., Okeene; Edward T. Shirley, M.D., Wynnewood; Carlton E. Smith, M.D., Henryetta.

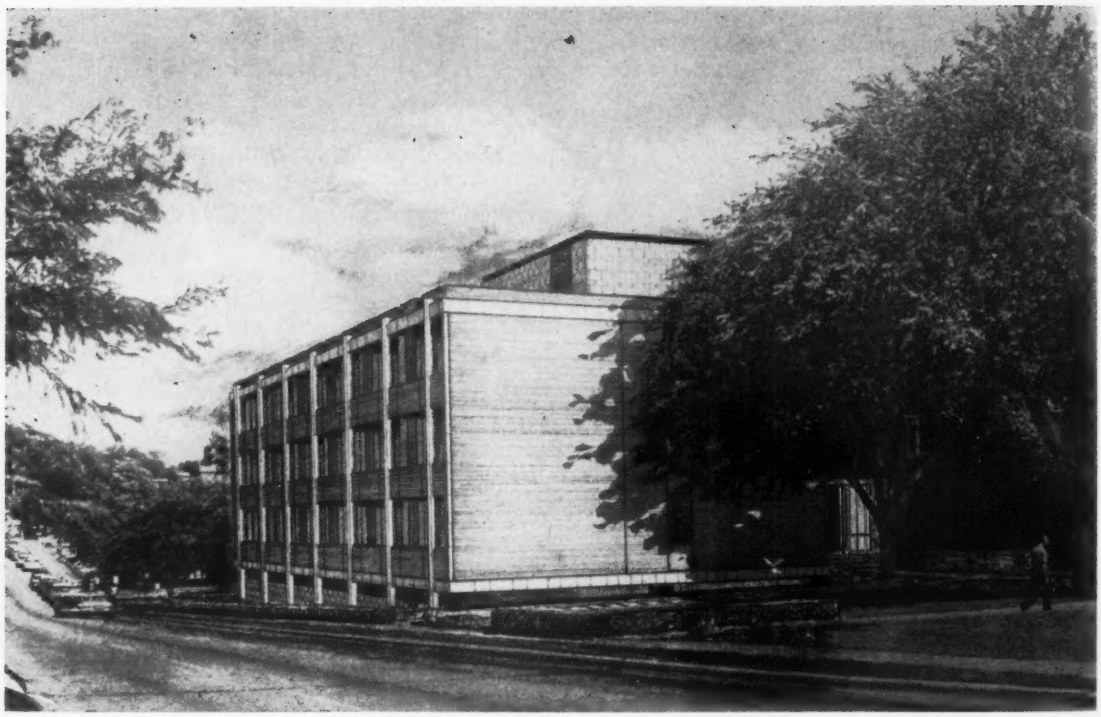
PHYSICIAN DISTRIBUTION

(Continued from Page 105)

verse relationship between this trend and that for physicians remaining from 1950 to 1959 in these same communities (Section A) suggests the explanation for these findings, i.e. the smaller the proportion remaining, the larger the new physician proportion is likely to be under these circumstances.

Section C (table II) demonstrates that the small and intermediate size communities with hospitals showed a significantly lower net loss of physicians, compared to their counterparts without hospitals. The lack of significant difference between communities of 2500 to 3500 population, with and without hospitals, points up the question as to whether or not the presence of a hospital per se is the primary factor influencing changes in physician population.

(To Be Continued)



Pictured above is the $800,000 expansion of the Oklahoma Medical Research Foundation. The building will be be constructed on the University Hospital property.

OU Medical Research Building Under Way

Groundbreaking ceremonies for the University of Oklahoma Medical Research Building were held January 5 under joint sponsorship of the Center and the Oklahoma City Chamber of Commerce.

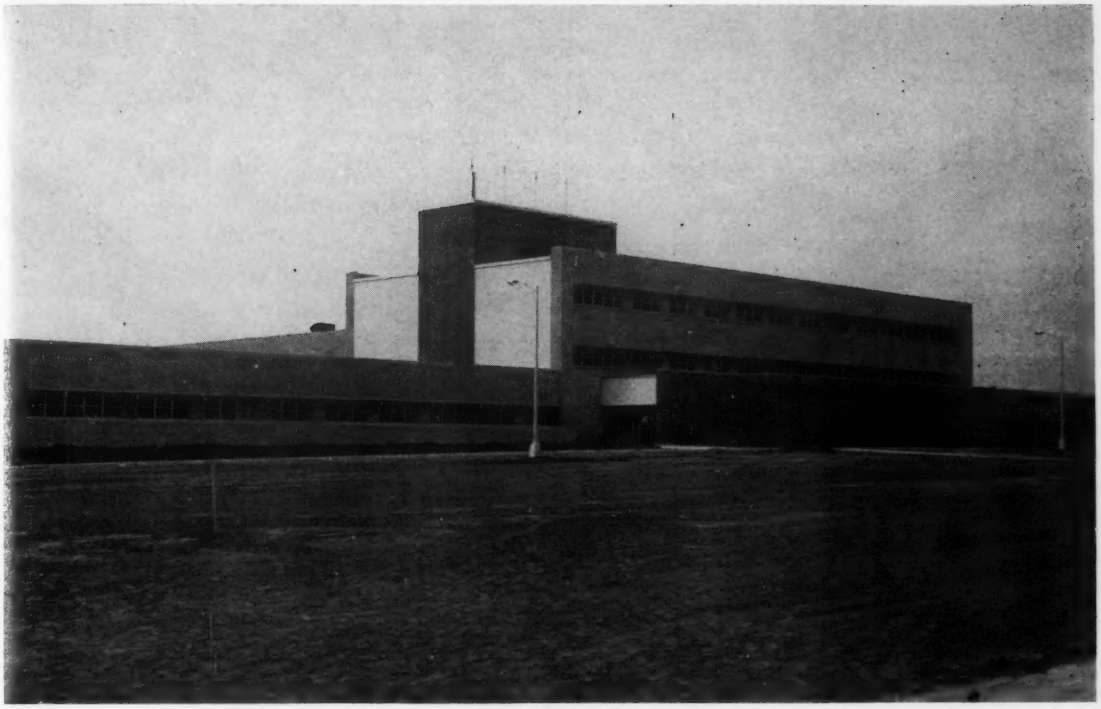
Paul A. Weiss, Ph.D., head of the Laboratory of Developmental Biology at the Rockefeller Institute for Medical Research, New York City, was principal speaker, addressing a luncheon at the Biltmore Hotel and speaking again at rites in the School of Medicine Auditorium. He came here as guest of Frontiers of Science Foundation.

Snowfall forced the actual groundbreaking indoors, where dignitaries performed the symbolic shoveling at a long wooden box filled with soil from the site. Bryce Baggett, Oklahoma City, state representative, presided at the brief program.

The approximately $800,000 building is being constructed on the south side of NE 13 Street in front of University Hospital and to the west of the hospital's Outpatient Clinics wing. The four-story structure will house some 50 laboratories and related facilities. University of Oklahoma Regents let the contract to Builders Construction Company of Oklahoma City at their December meeting.

Construction of much-needed laboratory space for faculty members was made possible by a $400,000 federal health research facilities grant awarded in 1957 and matched by a $400,000 appropriation of the 1959 state legislature.

Other speakers at the double-barrelled program included Lieut. Gov. George Nigh, George L. Cross, Ph.D., University of Oklahoma president, and Mark R. Everett, Ph.D., dean and director of the Center. Truett Coston, architect, was master of ceremonies at the luncheon.



Front view of Tinker's $2,368,962 modern hospital and dental clinic which was dedicated Saturday, January 23 by Dudley C. Sharp, Secretary of the Air Force.

New Tinker Air Force Base Hospital Opened

Formal dedication ceremonies were held January 23 for the new Tinker Air Force Base Hospital in Oklahoma City. Principle speaker for the occasion was Dudley C. Sharp, Secretary of the Air Force.

Covering 11 acres of the new area donated to the Air Force by the Oklahoma City Chamber of Commerce, the hospital is the first Air Force-designed hospital and dental clinic in the Air Force Medical Service.

The 75-bed fireproof hospital, with a 150-bed expansible plan, was built at a cost of $2,368,962. Construction was begun in May 1958 and completed in November 1959. It features complete air conditioning, with a thermostat in every room. There is piped-in central oxygen and vacuum to each patient area, a music distribution system with outlets to each patient's bed and nurses radio communication system to each patient. All patient bedrooms are semi-private with private baths and there are telephone and television outlets by each bed.

The east wing of the hospital houses the dental clinic which contains six dental operating rooms and two oral surgery rooms.

Three major operating rooms, an orthopedic plaster and splint room, sterile supply room and a post-surgical recovery room comprise the surgical suite.

Offices for the director, base medical services and the executive are located in the administrative wing. Also included in this wing are the business office, medical library and staff conference room.

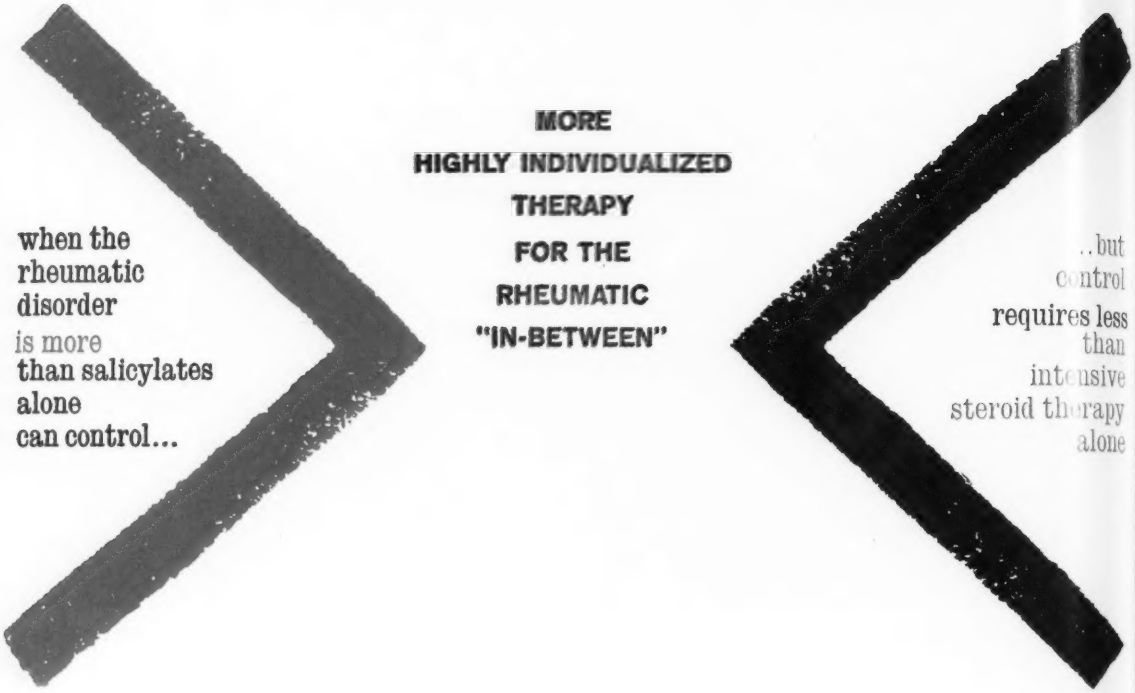
The modern structure, with its terrazo, asphalt and vinyl asbestos tile floors, mosaic ceramic-walled lobby, modern coved lighting, hi-fi music speakers, acoustical ceilings and hospital exchange is a valuable addition to the permanent Air Force facilities at Tinker Air Force Base.

Architects for the structure were Coston, Frankfurt and Short, Oklahoma City.

MORE
HIGHLY INDIVIDUALIZED
THERAPY
FOR THE
RHEUMATIC
"IN-BETWEEN"

when the
rheumatic
disorder
is more
than salicylates
alone
can control...

..but
control
requires less
than
intensive
steroid therapy
alone



Aristo

wider latitude in adjusting dosage

ARISTOGESIC is particularly effective for relief of chronic — but less severe — pain of rheumatic origin. ARISTOGESIC combines the anti-inflammatory effects of ARISTOCORT® Triamcinolone with the analgesic action of salicylamide, a highly potent salicylate. Dosage requirements for ARISTOGESIC are substantially lower than generally required for each agent alone. The exceptionally wide latitude of dosage adjustment with ARISTOGESIC permits well-tolerated therapy for long periods of time with fewer side effects.

Indications: Mild cases of rheumatoid arthritis, tenosynovitis, synovitis, bursitis, mild spondylitis, myositis, fibrositis, neuritis, and certain muscular strains.

Dosage: Average initial dosage: 2 capsules 3 or 4 times daily. Maintenance dosage to be adjusted according to response.

Precautions: All precautions and contraindications traditional to corticosteroid therapy should be observed. The amount of drug used should be carefully adjusted to the lowest dosage which will suppress symptoms. Discontinuance of therapy must be carried out gradually after patients have been on steroids for prolonged periods.

Each ARISTOGESIC Capsule contains:

ARISTOCORT® Triamcinolone	0.5 mg.
Salicylamide	325 mg.
Dried Aluminum Hydroxide Gel	75 mg.
Ascorbic Acid	20 mg.

Supply: Bottles of 100 and 1,000.

Lederle

RLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Have something to exhibit

If so, you should apply now for space at the 1960 meeting of your association. Scientific Exhibits will again play a major role . . . so why not prepare a graphic presentation of your work and share it with the 700 physicians who will attend the state's largest medical meeting? A research or clinical project may be shown as an individual exhibit, or you may present a single case in the "Klinical Kaleidoscope" section.

Research or clinical project

Perhaps you have done some original research, undertaken some experimental work, or simply drawn some conclusions based upon a series of interesting cases. Then you would be eligible to apply for an exhibit booth of your own in the Scientific Exhibit Section. A subject which has a special visual interest would be particularly suitable . . . and it need not be large—just stimulating!

Bizarre case

All of us have encountered a unique case which is worth talking about. If you have a well-documented one which you would like to share with others, toss your hat in the ring today by completing the application on the opposite page. The "Klinical Kaleidoscope" section will be a varied selection of bizarre cases, each presented with one to five x-rays or illustrations and accompanied by a very brief abstract of the clinical course.

O.S.M.A. ANNUAL MEETING

municipal auditorium · oklahoma city

may 2·3·4, 1960

APPLICATION FOR SCIENTIFIC EXHIBIT SPACE

54th Annual Meeting

OKLAHOMA STATE MEDICAL ASSOCIATION

Municipal Auditorium — Oklahoma City — May 2, 3, 4, 1960

1. **NAME AND ADDRESS OF APPLICANT:**

2. **TITLE OF EXHIBIT:**

3. **INDIVIDUAL BOOTH?** ☐ **"KLINICAL KALEIDOSCOPE" SECTION?** ☐

4. **SPACE REQUIRED:**

   A. **BOOTH: Indicate width in multiples of eight feet** _____
   (All booths will be 6 feet in depth)

   B. **"KLINICAL KALEIDOSCOPE": Indicate square footage:**
   Wall-type display _____ **Table top** _____

5. A. **GENERAL DESCRIPTION:**

   B. **MATERIAL TO BE SHOWN (Subject matter):**

   C. **METHOD OF PRESENTATION (x-ray, photos, graphs, models, etc.):**

6. **PREVIOUS SHOWINGS**

ACT NOW — SPACE IS LIMITED

Applications Must Be Received Prior to March 1, 1960

Linkletter Honors GP of the Year

The American Medical Association's GP of the Year, Chesley M. Martin, M.D., Elgin, Oklahoma appeared as a special guest on Art Linkletter's House Party, December 4, CBS-TV. Pictured above are the Martins with their host Mr. Linkletter.

What's Your Hobby, Doctor?

The DOCTOR'S HOBBY SHOW has become one of the outstanding attractions at the OSMA ANNUAL MEETING. A project of the Woman's Auxiliary, the show offers physicians an excellent opportunity to display the products of their leisure time. If you have a hobby, don't keep it a secret . . . Show your colleagues what you can do . . . APPLY NOW!

Doctor's Hobby Show

O.S.M.A. Annual Meeting

Municipal Auditorium

Oklahoma City

MAY 2, 3, 4, 1960

Application For Hobby Show Space

54th ANNUAL MEETING

OKLAHOMA STATE MEDICAL ASSOCIATION

DESCRIBE EXHIBIT, including information as to size, shape and value (insurance is provided):

IMPORTANT: Deliver Exhibit to Zebra Room, Municipal Auditorium by noon, May 1. Your Exhibit will be personally attended and insured at all times. It must be picked up by noon, May 4, when management responsibility ends.

MAIL THIS FORM TODAY!

State Chairman—Mrs. William R. R. Loney
2440 E. 26th Place
Tulsa 14, Oklahoma

State Co-Chairman—Mrs. Harrell C. Dodson, Jr.
1108 N.E. 42nd Street
Oklahoma City, Oklahoma

Course on Cardiac Patient Held

Four guest lecturers participated in a postgraduate short course on rehabilitation of the cardiac patient sponsored by the Oklahoma City Internist Association and Oklahoma State Heart Association February 10 at the University of Oklahoma Medical Center.

They were: Carlton B. Chapman. M.D., professor of medicine, Southwestern Medical School, Dallas; Amasa B. Ford, M.D., director of Rehabilitation Teaching Program, Western Reserve University, Cleveland; L. E. January, M.D., professor of medicine, State University of Iowa, Iowa City; Frederick J. Kottke, M.D., professor and head of the Department of Physical Medicine and Rehabilitation, University of Minnesota Medical School, Minneapolis.

The short course was combined with the Internist Association's annual Washington Birthday Clinic.

Doctor Chapman spoke on cardiovascular response to exercise and gave a second lecture on exercise in patients with mitral valve disease. Doctor Ford discussed various aspects of the relationship of physical activity to heart disease.

Problems in management of agricultural workers with heart disease were outlined by Doctor January. Doctor Kottke's subjects were "Cardiac Demands of Hospital and Everyday Activities" and "Planning a Rehabilitation Program for the Cardiac Patient."

Sessions began at 1 p.m. and 7 p.m. in the Medical School Auditorium. Seven faculty members took part. Guest participants included Judge Marx Childers, presiding judge, Oklahoma State Industrial Commission; Camp S. Huntington, M.D., assistant medical director, Phillips Petroleum Co.; Voyle Scurlock, director, Division of Vocational Rehabilitation, State Board of Education; and C. S. Lewis Jr., M.D., Tulsa, president, Oklahoma State Heart Association.

25 YEARS AGO

Articles published in *The Journal* of the Oklahoma State Medical Association, February, 1935.

DIABETIC PROBLEMS

Lea A. Reily, M.D.
Professor of Clinical Medicine, University of Oklahoma
Oklahoma City

Sir William Gull, Queen Victoria's physician, said: "What sin has Pavy committed or his father before him that he should be condemned to spend his life seeking a cure for an incurable disease?" Thus now we are sitting almost within the shadow of the laboratory where a definite treatment has been found lengthening the span of life so afflicted. This has been the outstanding development in medicine for the last decade. That, together with the fact that we are saving our children from the acute sudden deaths and condemning them to old age and diabetes. The high nervous strain of modern life with the excessive eating, has brought diabetes to morbidity and mortality. It is estimated in the states that there are one million diabetics, and that the mortality has raised from 20th to 10th place as the cause of death.

Mark Twain facetiously put it, "The way to attain longevity is to have a chronic disease and continually treat it." Joslin dedicated his 1934 Manual "To those individuals who have conquered diabetes by living longer with it than they were expected to live without it." Earlier diagnosis and better treatment as in tuberculosis, result in cases of longer duration and in compliance with the above, extends life and gives more chances for complications. The present diabetic is half and half a medical and surgical diabetic. Surgery is becoming more and more an increasing object of importance in handling those so afflicted, and the surgeon is faced with the care of a group of cases requiring special consideration, and leans on the internist for his advice and cooperation.

So we have direct and indirect complications in diabetes which touch all specialties in medicine, but I would liken the relationship of John Saxes' poem, where he described the Seven Blind Men of Hindoostan, who were wo wondrous wise, who went to see the elephant although none of them had eyes. Each saw the elephant from his own viewpoint, each one had a perception, but it takes conception of the diabetic in the form of some one physician who is in complete charge of the case in a medical ward who treats the diabetes, acting as the brains of the elephant who not only sees and governs the body as a whole, but who is not unmindful of the different members, remembering that diabetes is as jealous of her stellar role as an opera singer, and must be handled as carefully.

Letters

Dear Doctor Payne:

I noted with interest in this last issue of Round-up mention of your co-authorship of a paper on Clinical Evaluation of Weak Analgesic Agents. This brings to my attention again some recent thoughts I have had about analgesics and also muscle relaxing drugs which are put on the market in large amounts with a good deal of high pressure salesmanship on the part of the pharmaceutical firms. I note that in the last few years there has been an increasing tendency to have these drugs tested on what often seems to be a highly uncritical basis by investigators who are frequently not working in a University or comparable medical center. The investigation then is written up and published in one or another of the innumerable medical journals and reprints of the articles are used as sales promotion material by the drug firms. I strongly feel that in evaluating medications where there is so much difficulty in offering clear-cut objective evidence of effect, or lack thereof, the double-blind method of testing should routinely be employed and little attention paid to results obtained in any other way. Of course, it is quite difficult to offer any method of controlling this flood of questionably scientific literature, but it does seem to me at least that the editors of journals should be urged to take a much more closely critical look at papers submitted to them for publication. Possibly the Medical Writers' Society would be a place where someone could sound off on this subject and perhaps thereby bring it to the attention of editors of some of the journals.

I am taking the liberty of sending a copy of this letter to Dr. Ben Nicholson since the idea I mention may possibly be of interest to him.

I would like very much to see a copy of your paper on analgesic agents whenever it is conveniently available.

Sincerely,
S. FULTON TOMPKINS, M.D.

Dr. Tompkin's criticism is well taken and we state journal editors have little defense. In justice to us, however, it must be understood that our job as editors is not just selecting papers for publication from a great number that are submitted but procuring papers to make a decent journal since very few are submitted. In addition, we have a responsibility to our membership and the J. O. S. M. A. has adopted a policy that if a member submits a paper it will be accepted unless completely hopeless. We will not, in anyway, discourage a member who wants to write or report his work. Our greatest sin is the granting of permission to obtain large numbers of reprints for promotional purposes. In this we have no defense nor should we try to develop one.—The Editors.

Dear Doctor Nicholson:

The Oklahoma Society of Anesthesiologists wishes to announce the establishment of a Clinical Anesthesia Study Committee. This committee is set up to serve members of our Society and the Oklahoma State Medical Association by reviewing anesthesia protocols submitted by any Surgeon or Anesthetist.

The Committee is composed of four Anesthesiologists each of whom will review the protocol and make comments and recommendations which will be forwarded to the person submitting the case.

An elaborate procedure has been set up to assure complete anonymity of the patient,

surgeon, and person administering the anesthesia in every case. The Secretary of the Oklahoma Society will be the only person knowing both the name of the person submitting a case and its number, and he will never see either the case report or the comments.

The Committee solicits cases for study from surgeons or their anesthetists in addition to the members of our Anesthesia Society. Blanks for submitting case reports may be obtained free by writing to the Secretary of the Oklahoma Society of Anesthesiologists, c/o Department of Anesthesia, University Medical Center, Oklahoma City, Oklahoma. The Committee reserves the right to publish interesting cases without identifying numbers or names in this *Journal* or other medical journals. Cases cannot be reviewed unless they are reported on the standard forms furnished by our Society.

Sincerely yours,
R. GIBSON PARRISH, M.D.

The Journal will publish the reviews that are submitted by the committee.

Have You Heard?

ROBERT H. FURMAN, M.D., director of cardiovascular research at the Oklahoma Medical Research Foundation, has been appointed to the heart study section of the National Institutes of Health, research arm of the U.S. Public Health Department. Members of the study section are investigators who review all applications for grants from the public health service to support research in heart disease.

WILLIAM W. SCHOTTSTAEDT, M.D., University of Oklahoma Medical Center faculty member since 1953, has been named chairman of the Department of Preventive Medicine and Public Health. He succeeds Kirk T. Mosley, M.D., associate dean in charge of special training and research programs.

LEO LOWBEER, M.D., pathologist at Hillcrest Medical Center, Tulsa, will be a lecturer at the 12th Annual Meeting of the American Academy of Forensic Sciences, March 3-5, in Chicago.

EVANS JAMES KURTS, M.D., is joining A. W. Haddox, M.D., as a partner in the Haddox Clinic in Antlers. Doctor Kurts is a graduate of the College of Medicine, University of Tennessee, Memphis, and has been taking a residency in surgery at Hillcrest Hospital in Tulsa.

C. A. TRAVERSE, M.D., Alva, has instigated a rather unique feature at an Alva hospital staff meeting. ART YOUNG, M.D., orthopedic specialist from Freeport, New York, discussed practical orthopedic problems with the group for one hour and fifteen minutes via amateur "ham" radio. The lecture proved so interesting and profitable that a similar "meeting" of the specialist and Alva physicians has been planned for a later date.

LYNN BARNES, M.D., Nowata physician, has been named local President of the Chamber of Commerce for 1960.

CHARLES W. LETCHER, M.D., Miami, retired from the Navy, has received a commission as Rear Admiral (Medical Corps) United States Naval Reserve.

ROBERT K. MCINTOSH, M.D., has been selected Outstanding Citizen of 1959 in Tahlequah. While most letters of recommendation for the honor mentioned a single area of service, one seemed to sum up the appreciation " . . . Doctor McIntosh has given his life to this community, commanding the highest respect in his profession. Tahlequah is lucky to have doctors like him."

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER
POSTGRADUATE PROGRAM*
Individual Postgraduate Courses

BASIC ELECTROCARDIOGRAPHY — February 29 through March 5.

OPTHALMOLOGY-OTOLARYNGOLOGY SYMPOSIUM—March 3 and 4.

OBSTETRICS-GYNECOLOGY SYMPOSIUM—March 5.

ADRENAL STEROIDS—March 24, 25, and 26. (Third Oklahoma Colloquy on advances in Medicine.)

ORTHOPEDIC SYMPOSIUM—May 13 and 14.

CARCINOMA OF THE SKIN—April 22 and 23. (Sixth Annual Combined Surgery, Radiology, Pathology Symposium.)

OKLAHOMA ASSSOCIATION OF HOUSE STAFF PHYSICIANS—May 6.

PEDIATRIC NEUROLOGY—May 12 and 13. This symposium to be held in conjunction with the Wednesday Short Course on May 11.

SERIAL POSTGRADUATE COURSE

Postgraduate Division

Oklahoma City, Oklahoma
1959-1960

Wednesday Short Courses
3:30 to 8:30 p.m.

Mar. 9	C. B. Taylor Lectureship and Urology Symposium—Guest Lecturer to be announced
Apr. 13	Anesthesia for the Part-time Anesthetist
May 11	Neurological Diseases in Childhood
June 8	Surgery

*Further information may be obtained from the Office of Postgraduate Education, University of Oklahoma School of Medicine, 801 N.E. 13th Street, Oklahoma City, Oklahoma.

OKLAHOMA CHAPTER

AMERICAN ACADEMY OF GENERAL PRACTICE

February 15-16, 1960 **Tulsa Hotel**
Tulsa, Oklahoma

The 12th Annual Meeting of the Oklahoma Chapter of the American Academy of General Practice will be held at the Tulsa Hotel, February 15-16, 1960, in Tulsa. Complete program on page 43.

UNIVERSITY OF TEXAS
POSTGRADUATE SCHOOL OF MEDICINE

February 25, 26, 27, 1960 **Houston 25, Texas**

The University of Texas Postgraduate School of Medicine is presenting a program on Gastroenterology in the Texas Medical Center, Houston, Texas on February 25, 26, 27, 1960. Emphasis on diagnosis and therapeutic approaches, medically and surgically, will be discussed.

For further information write: Office of the Dean, The University of Texas Postgraduate School of Medicine, 410 Jesse Jones Library Building, Texas Medical Center, Houston 25, Texas.

TEXAS POSTGRADUATE SCHOOL OF MEDICINE

Fifth Annual Course in Anesthesiology

March 9, 10 ,11, 1960 **Houston, Texas**

The University of Texas Postgraduate School of Medicine announces the Fifth Annual Course in Anesthesiology to be held March 9, 10, 11, 1960 in Houston. The course is designed to review theory and practice of commonly used anesthetic techniques. Inquiries may be addressed to The University of Texas, Postgraduate School of Medicine, 410 Jesse Jones Library Building, Texas Medical Center, Houston 25, Texas.

SCOTT AND WHITE CLINIC
EIGHTH ANNUAL POSTGRADUATE CONFERENCE

March 6, 7, 8, 1960 **Temple, Texas**

This course, Current Topics in Medicine and Surgery, will be presented by Scott, Sherwood and Brindley Foundation and the University of Texas Postgraduate School of Medicine on March 6, 7, 8, 1960. Registration fee is $25.00 and the course is approved for 20 hours credit by AAPG. Further information is available by writing to Publications Office, Scott and White Clinic, Temple, Texas.

INSTITUTE IN PSYCHIATRY AND NEUROLOGY

February 25-26, 1960 **Little Rock, Arkansas**

The Twelfth Annual Institute in Psychiatry and Neurology will be held at the Consolidated VA Hospital, North Little Rock Division, Little Rock, Arkansas, on February 25-26, 1960. Additional information may be obtained by writing H. B. Witten, M.D., Assistant Director of Professional Services for Educaion, North Little Rock Division, Little Rock, Arkansas.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

March 7-10, 1960 **Roosevelt Hotel**
New Orleans, Louisiana

The twenty-third annual meeting of The New Orleans Graduate Medical Assembly will be held March 7-10, 1960 in New Orleans. Complete details may be obtained by writing to Mannie D. Paine, Jr., M.D., Secretary, The New Orleans Graduate Medical Assembly, 1430 Tulane Avenue, New Orleans 12, Louisiana.

Auxiliary News

25th Annual Doctors' Day

Definite planning has begun in county auxiliaries throughout southern states for our 25th annual observance of Doctors' Day. In fact, our *Sooner Physician's Wife*, through its excellent coverage of Doctors' Day, has helped stimulate interest in many states not included in the Southern Medical Association.

March 30 is the anniversary of the pioneer use of ether by Doctor Crawford W. Long—1842 was the year; Jefferson, Georgia, the place. In 1935 the first observation was held by a medical auxiliary. (Why not refresh your memory and re-read the interesting account in Fülöp-Miller's *Triumph Over Pain?* Do you remember the ether parties that led to Doctor Long's conclusions, eventual ostracism, and the dramatic conclusion?)

The variety of ways in which we celebrate March 30 is endless: with proclamations, red carnations, recognition in church services, and so on. The entertainments planned vary from formal Carnation Balls, with proceeds going to memorials, to chuck-wagon dinners. Some celebrate with solemnity, some with frivolity; regardless of manner, it is an occasion we all anticipate.

Our State Doctors' Day Chairman is Mrs. Lawrence E. Thompson, 4041 South Richmond, Tulsa.—And lest you forget, Mrs. Pat Fite, Sr., 1417 Emporia, Muskogee, is our State A.M.E.F. Chairman. What more timely way is there to thank our own family doctors than to make a contribution to A.M.E.F. in their honor?

Futures, Inc.

It is also timely, both in personal and organizational planning, to give much thought to strengthening our Paramedical Careers program. One auxiliary is planning a tea for the officers and sponsors of Future Nurses' Clubs in order to give them an opportunity to exchange ideas and evaluate their programs. Mrs. H. C. Manning, 1025 East Broadway, Cushing, is our Paramedical Recruitment Chairman, and has information available to answer any of your questions.

Meetings held before State Future Nurses' Day, March 19, should be geared to produce interest in attendance at the state meeting; those following this date should be of evaluation nature.

Auxiliaries should especially stress the availability of our Nurse Loan Fund program to every high school in the state, regardless of contact through organized clubs. Upon acceptance of any girl at any qualified school of nursing, application to our Nurse Loan Fund can be made immediately. Familiarity with the availability of this Fund may be the decisive factor in the plans of many spring graduates; and it cannot be over-emphasized. Mrs. William R. Cheatwood, Box 870, Duncan, is State Loan Fund Chairman.

AMERICAN COLLEGE OF ALLERGISTS

Graduate Instructional Course and Annual Congress

February 28-March 4, 1960 **The Americana Hotel**

Bal Harbor

Miami Beach, Florida

The American College of Allergists will hold a Graduate Instructional Course and Annual Congress February 28 to March 4, 1960 at the Americana Hotel, Bal Harbor, Miami Beach, Florida. For further information, contact John D. Gillaspie, M.D., Treasurer, 2049 Broadway, Boulder, Colorado.

PAN-PACIFIC SURGICAL ASSOCIATION

EIGHTH CONGRESS

September 27-October 5, 1960 **Honolulu, Hawaii**

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 27 through October 5, 1960. All members of the profession are eligible to register.

Further information and brochures may be obtained by writing to F. J. Pinkerton, M.D., Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

PHYSICIAN PLACEMENT

Dermatology

Thomas E. Slimp, M.D., 323 West Livingston Place, Metairie, Louisiana, age 42, married, Syracuse Medical School, 1950, board certified January 1, 1960. No military obligations, available January 1, 1960.

Gordon Harold Ekblad, M.D., Field Medical Service School, Camp Pendleton, California, 53, married, University of Minnesota, 1930, available January 1960.

General Practice

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

D. H. Bessesen, M.D., Grand Canyon Hospital, Grand Canyon, Arizona, age 61, married, University of Minnesota, interested University Health Service, available October 1, 1959.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Paul J. Vega, M.D., 6070 Orleans Ave., New Orleans, Louisiana, age 26, married, Louisiana State University, 1956, no military obligations, available now.

Thomas S. Whitecloud, M.D., 858 Market Street, Pascagoula, Mississippi, age 45, married, Tulane, 1943, veteran, has interest in teaching, availability date depends on situation.

Internal Medicine

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Albert D. Roberts, Jr., M.D., 4333 Colgate, Dallas, Texas, 29, married, Southwestern Medical School, Veteran, available July 1959.

Van B. Saye, Jr., M.D., 1230 Brockenbraugh Court, Metairie, Louisiana, age 28, married, Medical College of Georgia, 1954, Air Force Reserve, available July 1, 1960.

Locum Tenens

Harold W. Calhoon, M.D., Wesley Hospital, Oklahoma City. One year residency in Internal Medicine. Available July 1 to September 15th, 1960.

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Hospital, Tulsa, desires two weeks' tenure between now and July 1. Write to St. John's or call RIverside 3-3059.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Lynn W. Abshere, M.D., 708 N.E. 14th, Oklahoma City, age 31, married, University of Oklahoma School of Medicine, 1956, American College of OB-GYN, no military obligations, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Milton Gardner Mutch, Jr., M.D., 423 Mills Avenue, Fargo, North Dakota, age 31, married, University of Minnesota, 1955, board eligible upon completion of fellowship, veteran, available July 1, 1959.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Alvon C. Winegar, M.D., 1940 Michigan, Benton Harbor, Michigan, age 41, married, Wayne University, 1942, no military obligation, available early 1960.

Deaths

HARLEY M. PRENTISS, M.D.
1892-1959

Harley M. Prentiss, M.D., 67-year-old Nowata physician, died in Rochester, Minnesota on December 26, 1959.

A native of Metamora, Ohio, Doctor Prentiss graduated from the University of Oklahoma School of Medicine in 1918. He established his practice in Yale, Oklahoma in 1920, later moving to Nowata and then to Roosevelt, Oklahoma. He returned to Nowata in 1948.

Doctor Prentiss was a Past-Secretary of the Washington-Nowata County Medical Society.

JACK W. MYERS, M.D.
1917-1960

Jack W. Myers, M.D., 43-year-old El Reno physician, died at his home on January 3, 1960.

Born in Apache, Oklahoma, Doctor Myers graduated from the University of Oklahoma School of Medicine in 1943. After taking a residency at the City-County Hospital in Fort Worth, Texas, he established his practice in El Reno.

Doctor Myers served in the army medical corps during World War II and with the air force in the Korean conflict.

JOHN REYNOLDS, M.D.
1874-1959

John Reynolds, pioneer Muskogee physician, died December 28, 1959 in Muskogee.

A native of Asheville, North Carolina, Doctor Reynolds graduated from the University of Louisville Medical College in 1903. All but one year of his 57-year medical career was spent in Muskogee.

In 1952, Doctor Reynolds was honored for his years of service to the medical profession when the Oklahoma State Medical Association presented him a 50-year pin.

WILLIAM A. WALKER, M.D.
1892-1959

William A. Walker, M.D., 67-year-old Tulsa physician, died December 31, 1959 in Tulsa.

Born in Holdenville, Doctor Walker graduated from the University of Oklahoma School of Medicine in 1927. After establishing his practice in Tulsa, he served as public schools physician for eight years.

Doctor Walker was a member of the Oklahoma Chapter, American Academy of General Practice.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Olin O. Williams, Jr., M.D., 3768 Alma Drive, Memphis, Tennessee, 29, married, University of Tennessee, veteran, available January 1962.

Urology

Charles Lee Reynolds, Jr., M.D., 5012 Rockport Drive, Dallas, Texas, age 32, Southwestern Medical College, Dallas, 1949, board eligible, married, no military obligations, available July 15, 1960.

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas, 1951, board certified, veteran, available now.

Herbert Sohn, M.D., 2010 Cliffview Road, Cleveland, Ohio, 32, married, Chicago Medical School, 1955, veteran, available July 1960.

MISCELLANEOUS ADVERTISEMENTS

FOR SALE: Autoclave, Castle, electric. Originally purchased for $1866.75 in 1956. Contact Walt Allen, 116 North Fourth Street, Chicaksha, Oklahoma.

FOR SALE or RENT: Office building, equipment and supplies, including x-ray. Residence also available. Contact Box F, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: General practice established 7 years in prosperous oil and agriculture community. Town is 5000 and trade area over 15,000 population. Gross receipts over $50,000 annually. New clinical building for sale or rent. New community hospital expected within a year. $30,000 for practice, equipment and current accounts receivable. Terms. Owner going into residency. Contact Box L, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

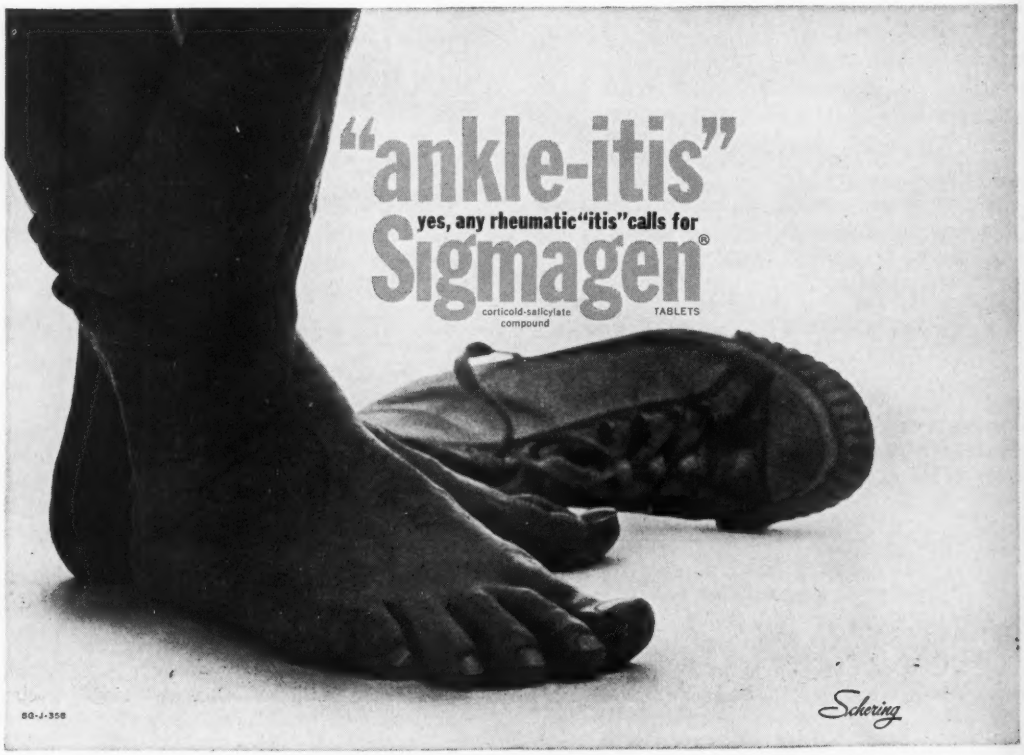
FOR SALE: $40,000 per year General Practice. 14 room completely equipped clinic. County seat town of 4,000 with new 40 bed hospital. Liberal terms. Write Key J, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

OFFICE SPACE for rent, modern building, 528 N.W. 12th, Oklahoma City. Complete maintenance, air conditioning, free parking space for patients. CE 6-1631.

WANTED: Internist to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR RENT: Office space for two doctors, modern, air conditioned building. 3832 N. May Ave., Oklahoma City. Complete maintenance. Free parking space for patients. WI 2-5531.

WANTED: Surgeon to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

WILL CUSTOM BUILD offices for physicians and dentists in new medical center located on North Western in Oklahoma City. Approximately 500 square feet per unit. Ample parking. Construction to start immediately. Contact Bob Fowler, P.O. Box 9554, Shartel Station, Oklahoma City, WI 2-1021.

WANTED: General Practitioner with some ability to do surgery. Primarily industrial practice. Beginning salary $900.00. Contact C. J. Paternostro, M.D., 426 Medical Arts Building, Dallas, Texas.